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Original Communications

COMPLETE REPLACEMENT OF THE MITRAL VALVE

Successful Clinical Application of a Flexible Polyurethane Prosthesis

Nina S. Braunwald, M.D., Theodore Cooper, M.D., Ph.D., and Andrew G. Morrow, M.D., Bethesda, Md.

INCREASING numbers of open operations for the correction of mitral regurgitation have been performed in recent years. The experience in this clinic, as well as at other centers, has shown that, in a large proportion of patients with mitral regurgitation, reconstructive procedures utilizing existing valve tissue have been followed almost universally by disappointing clinical and hemodynamic benefit at the expense of significant operative risk. The poor results of reconstructive operations are largely related to the extensive immobility, calcification and loss of valvular substance which is encountered, and the need for a suitable prosthesis for complete replacement of the mitral valve has become increasingly evident. Many types of prosthetic mitral valves, constructed of either rigid or flexible materials, have been designed and evaluated in experimental animals[1, 2] and a few reports of clinical application have been made.[3, 4] To our knowledge, however, complete excision and successful replacement of the mitral valve has not been previously described. In the present report the design and methods of fabrication of a prosthetic valve constructed of flexible polyurethane foam are presented and the application of this valve in the surgical treatment of 2 patients with mitral regurgitation is described.

DESIGN, FABRICATION, AND EXPERIMENTAL EVALUATION

In considering the mechanical design of the valve it was thought that a prosthesis which closely approximated the anatomy of the normal mitral valve

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would be most likely to provide an effective functional substitute. Such a design takes advantage of ventricular contraction as well as the pressure differences between the atrium and ventricle. Wax or plaster casts were made of the mitral ring and the mitral valve leaflets in fresh normal or diseased human hearts and in the hearts of normal dogs and calves (Fig. 1). The casts were trimmed and smoothed and from them two-piece molds were made of Devcon, an industrial molding compound* (Fig. 2). In constructing the valve, a thin piece of knitted Dacron fabric was stretched over the male half of the mold and secured around

Fig. 1.—Plaster *(left)* and wax *(right)* casts of the mitral valve made at autopsy.

Fig. 2.—A completed mold in which the valve is fabricated.

its base. The female half of the mold was then half-filled with catalyzed liquid polyurethane† and the mold was quickly closed and compressed. The valve was cured in the mold overnight, then removed and trimmed. The valve orifice was cut and "chordae tendineae" of Teflon tape were sutured to the leaflets as shown in Fig. 3.

Valves constructed in the manner described were used for complete replacement of the mitral valve in 27 dogs. A left thoracotomy was made since it was found that the small left atrium of the normal dog could be incised more widely from this approach. After cannulation of the jugular and femoral veins

*Manufactured by the Devcon Corporation, Danvers, Mass.
†Manufactured by American Latex Company, Hawthorne, Calif.

and the femoral artery, complete cardiopulmonary bypass was instituted, the left atrium was opened widely and the mitral valve was excised. The prosthesis was then inserted in the manner described and illustrated later. After the atriotomy had been closed and bypass discontinued, the competency of the valve was assessed by measurements of left atrial pressure and the tension on the chordae was adjusted (Fig. 4). A mean left atrial pressure of 8 to 12 mm. Hg

Fig. 3.—The completed valve before *(left)* and after *(right)* incision of the commissures and attachment of the chordae.

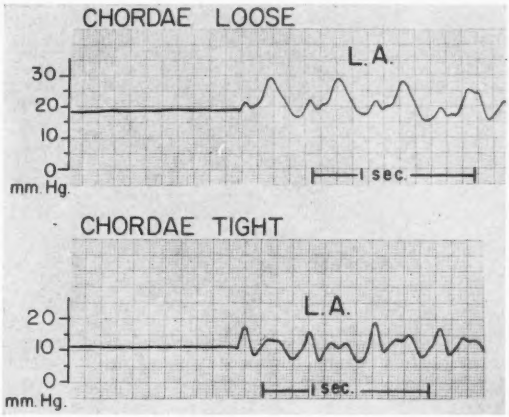


Fig. 4.—Left atrial pressure tracings obtained in an experimental animal before and after adjustment of the tension on the chordae of the prosthetic valve.

was considered evidence of satisfactory valve function when systemic arterial pressure was normal, and this level of atrial pressure was achieved in six of the last ten experiments.

In the initial experiments no systematic attempt was made to obtain survival since not only were valves of various designs employed but the technique of insertion was also modified continually. In more recent operations, when the design of the valve and surgical technique had been standardized, chronic survival was still not achieved but 4 dogs lived from 8 to 40 hours. In them, autopsy revealed that the valve surface was covered with a thin layer of fibrin but clot formation was never observed; there was no evidence that death

resulted from mitral regurgitation or stenosis since pulmonary edema was not evident. In the entire experimental experience it was thought that death was principally related to the technical difficulties of suturing the valve into the normally small and thin-walled atrium of the dog. In several instances the sutures tore the annulus and massive bleeding ensued; in other experiments the animals died of the sequelae of prolonged perfusion.

In spite of the difficulties described, it was considered that the basic objective, a satisfactory prosthetic valve, had been achieved and experience with reconstructive operations indicated that the technical problems of its insertion in patients would be far simpler than in the normal dog.

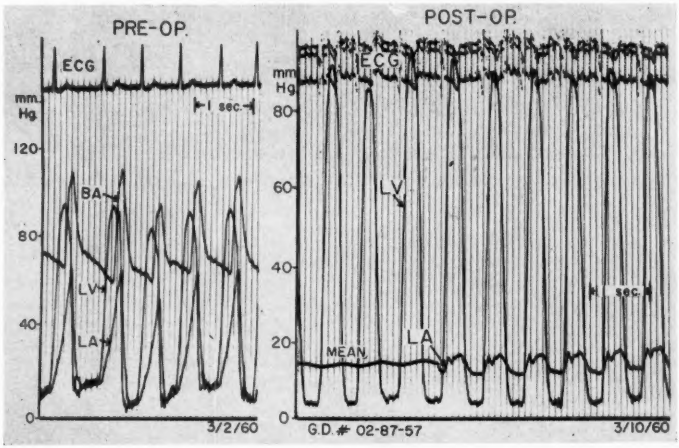


Fig. 5.—Records of left atrial *(LA)*, left ventricular *(LV)*, and brachial arterial *(BA)* pressures obtained in patient G. D. at preoperative catheterization *(left)* and after insertion of the prosthetic valve *(right)*.

CLINICAL APPLICATION

The prosthesis described has been employed in 2 patients for complete mitral valve replacement.

CASE 1.—G. D. was a 16-year-old schoolgirl in whom no history of rheumatic fever was obtained. She had been relatively well until 2 years before admission when she began to experience dyspnea, fatigability, and ankle edema. All of these symptoms progressed and she had been hospitalized on several occasions in congestive heart failure. There was a prominent left ventricular lift and a systolic thrill at the apex. A Grade 4/6 systolic murmur was audible over the whole precordium, maximal at the apex and referred to the axilla. No diastolic murmur was present. The chest roentgenogram showed gross cardiac enlargement with prominence of the left ventricle and a greatly dilated left atrium. The electrocardiogram revealed atrial fibrillation.

Combined right and transseptal left heart catheterization showed the right ventricular pressure to be elevated (52/10 mm. Hg). The Kr^{85} test of 2.6 per cent in this chamber excluded a left-to-right shunt. The mean left atrial pressure was 27 mm. Hg and peaks of the v waves were 50 to 60 mm. Hg. There was no diastolic gradient across the mitral valve (Fig. 5).

At operation (March 10, 1960) the anterior leaflet of the mitral valve was found to be contracted, deformed, and extensively scarred. Both leaflets were excised and the prosthesis inserted. Bleeding from a tear in the left atrial suture line necessitated prolonged partial cardiopulmonary bypass until it could be controlled. At the conclusion of the procedure, simultaneous measurements revealed a satisfactory reduction in mean left atrial pressure and a small mitral diastolic gradient (Fig. 5). Palpation of the functioning valve revealed it to be mobile and only a slight regurgitant jet could be felt, apparently localized to the posterior commissure.

Postoperatively the patient reacted promptly, was alert and maintained a normal blood pressure without support. The cardiac rhythm spontaneously reverted to a sinus mechanism. Only a faintly audible systolic murmur was present. After the first 2 hours, however, she became oliguric and the serum potassium, in spite of treatment, rose to 7.3 mEq. per liter. She suddenly became hypotensive and died 60 hours after operation.

At autopsy the valve was found to be in good position and its atrial surface showed no fibrin or clot. Inspection from the ventricular aspect revealed that in placement of the artificial chordae, one had inadvertently been passed behind the other. This had resulted in obstruction to the valve opening where the chordae crossed. A fibrin clot occupied this area.

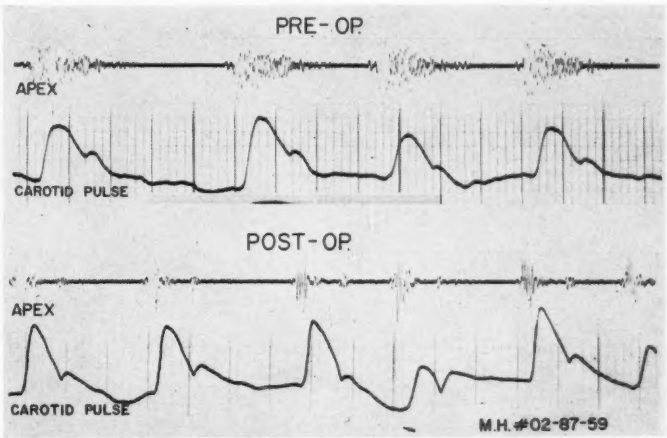


Fig. 6.—Phonocardiograms recorded at the apex of the heart in patient M. H. before and after operation. No systolic murmur is audible or recorded after insertion of the prosthesis.

CASE 2.—M. H. is a 44-year-old housewife in whom a heart murmur and cardiac enlargement had been noted since the age of 8 years. There was no history of rheumatic fever. She had had dyspnea and fatigability for many years and since a pregnancy in 1943 all her symptoms had progressed. Six months before admission, congestive failure, edema, orthopnea, and pleural effusion had necessitated hospitalization.

She was thin and appeared chronically ill. The neck veins were dilated, the blood pressure was 110/70 mm. Hg and the pulse completely irregular at a rate of 70 to 80. Numerous crackling râles were heard at both lung bases. The heart was enlarged, the apical impulse lying in the seventh intercostal space outside the anterior axillary line. Both left and right ventricular lifts were palpable and a systolic thrill was present at the apex. The pulmonary second sound was increased but normally split. A Grade 5/6 pansystolic murmur was present at the apex and radiated to the axilla (Fig. 6). A Grade 2/6 diastolic murmur was heard in this area also. The edge of the liver was palpable 6 cm. below the right costal margin. The chest roentgenogram (Fig. 7) demonstrated

gross cardiac enlargement with prominence of the left ventricle, left atrium, and pulmonary artery. There was considerable pulmonary congestion. The electrocardiogram showed atrial fibrillation, right axis deviation, and nonspecific S-T segment and T-wave changes, as well as digitalis effect.

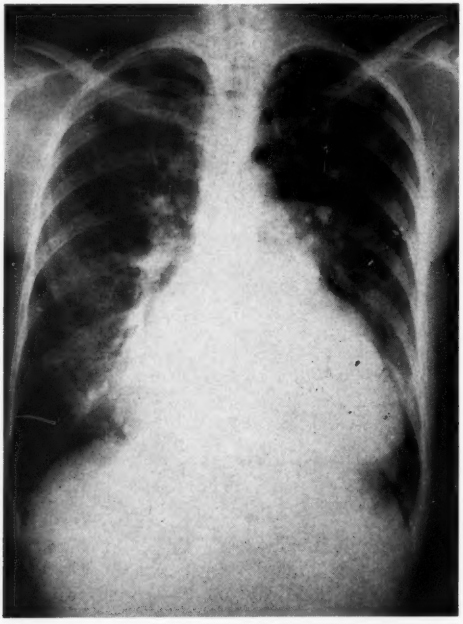


Fig. 7.—Preoperative roentgenographic appearance of heart in patient M. H.

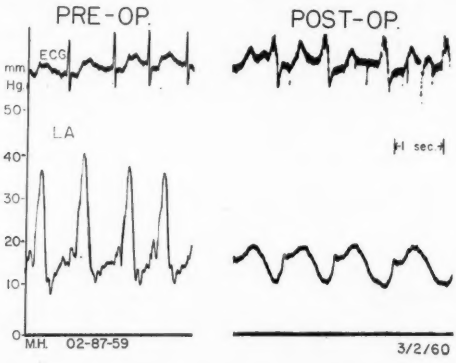


Fig. 8.—Records of left atrial pressure obtained at preoperative catheterization *(left)* and after the insertion of the prosthesis *(right)* in patient M. H.

At right heart catheterization the right ventricular pressure was elevated to 55/10 mm. Hg and the right atrial mean pressure was 11 mm. Hg. Unexpectedly, a small left-to-right shunt entering the right atrium was demonstrated by inhaled Kr^{85} tests. The catheter was then passed across an interatrial communication and into the left ventricle. Arterial indicator dilution curves recorded after left ventricular injection demonstrated

a prolonged descent compatible with valvular regurgitation. The left atrial mean pressure was 20 mm. Hg and the pulse contour was that of gross mitral regurgitation (Fig. 8). There was no diastolic gradient across the mitral valve. When the catheter was withdrawn across the interatrial septum a distinct pressure gradient between the left and right atria was recorded.

It was considered that the patient had either an incomplete form of persistent atrioventricular canal or a small atrial septal defect of the usual type with rheumatic mitral regurgitation. At operation (March 11, 1960) palpation of the interatrial septum revealed a small defect in the region of the foramen ovale. Palpation within the left atrium disclosed gross mitral regurgitation through a scarred and thickened valve. There was no calcification. The valve lesion was considered to be of rheumatic rather than of congenital origin. A left atriotomy was made and the anterior leaflet was seen to be curled, thickened, and scarred; the posterior leaflet was rolled and immobile. Both leaflets and their attachments to the papillary muscles were excised. The chordae of the prosthetic valve were placed and the valve sutured to the thickened annulus. The interatrial septal defect, which was 1.5 cm. in diameter, was closed with several interrupted sutures. When left ventricular output had been restored the chordae were adjusted in length until no regurgitant jet could be felt. Pressure measurements after the repair (Fig. 8) showed a small diastolic gradient across the valve but the pulse contour characteristic of mitral regurgitation was absent.

Postoperatively an elective tracheostomy was performed. The patient's blood pressure was supported by small doses of Aramine for 24 hours, after which this was unnecessary. She has made a slow but uneventful recovery and is apparently well 6 weeks after operation. No precordial murmur is audible (Fig. 6).

SURGICAL TECHNIQUE

The operative method employed in each patient was similar. The patient is positioned supine with the right chest elevated to 30-40 degrees and a bilateral anterior thoracotomy is made extending over the right fifth intercostal space and toward the apex of the heart in the left sixth interspace (Fig. 9). Tapes are passed about the venae cavae and the aorta freed from the pulmonary artery to permit occlusion if necessary. The anterior wall of the left atrium is dissected from the right atrium if a large area of it is not evident. After cannulation of the venae cavae and femoral artery, bypass is instituted and a left atriotomy made (Fig. 10). The mitral valve is grasped and excised from the papillary muscles and annulus; a rim of valve tissue, 2 to 3 mm. in width, is left attached to the annulus. The anterior chorda of the prosthetic valve is threaded to a Reverdin needle and passed from the cavity of the left ventricle through its wall near the base of the anterior papillary muscle, the point of the needle being directed so that major coronary vessels are avoided (Fig. 11). The posterior chorda is similarly inserted through the ventricular wall near the base of the posterior papillary muscle. Each chorda is held with a clamp and a mattress suture of heavy silk placed and tied about the site of exit from the heart. The valve is then drawn into position and its commissures carefully aligned as the natural ones were. The free margin of the valve is then anchored with mattress sutures placed in four quadrants of the annulus and the fixation completed with continuous sutures between these points (Fig. 12). After the air has been evacuated from the heart the left atrium is closed with a continuous everting mattress suture except for an area in the center of the suture line

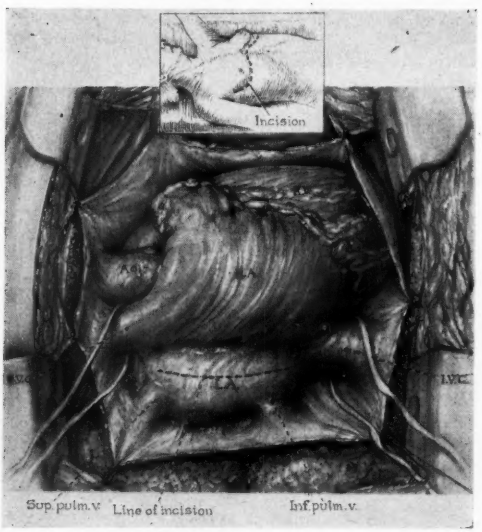


Fig. 9.—Operative exposure employed in mitral valve replacement.

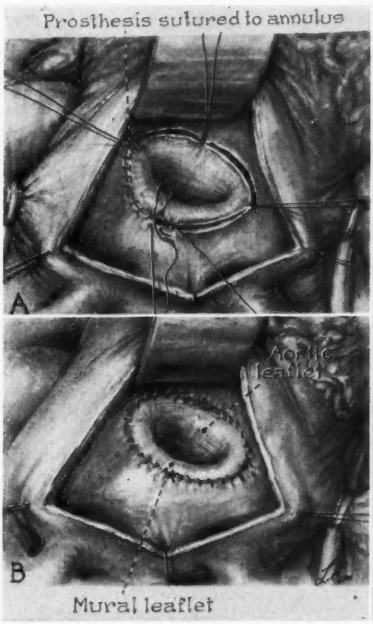
Fig. 10.

Fig. 11.

Fig. 10.—After total cardiopulmonary bypass has been established the valve is exposed and excised as shown.

Fig. 11.—Methods by which the artificial chordae tendineae are placed and later secured to the left ventricular wall.

where interrupted sutures are placed but not tied. The finger is inserted through this opening and the vena cava tourniquets are loosened, permitting approximately half the systemic venous return to enter the heart. The length of each chorda is then adjusted until, by palpation, the valve is made maximally competent. They are fixed in this position to the left ventricular wall, buttressed by squares of Teflon felt. The final position of the prosthesis is shown in Fig. 13.



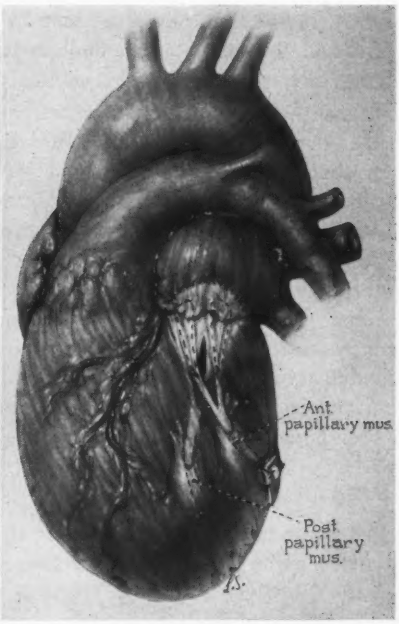


Fig. 12. Fig. 13.

Fig. 12.—Fixation of the prosthesis to the mitral annulus.
Fig. 13.—Final position and appearance of the prosthesis after insertion.

COMMENT

In the design of the prosthetic valve described, it was considered that the degree of flexibility of the leaflets was of prime importance. When they were made too rigid not only was a relatively high pressure required to open the valve, but there was sufficient resistance to closure in systole so that regurgitation occurred. The extreme flexibility finally achieved precluded any form of internal fixation to prevent leaflet eversion and necessitated the artificial chordae tendineae employed. Early experience with the valve indicated that the length of the chordae was critical and that it was difficult if not impossible to correctly estimate their optimal length when they were sutured to the papillary muscles

or ventricle before the valve itself was inserted and functioning. For this reason the chordae were brought through the ventricular wall, adjusted and fixed in the manner described. This technique permits regulation of the tension on the chordae when the ventricle is effectively contracting and, by palpation, optimum competency can be obtained.

Many plastic materials were evaluated in the fabrication of valves. The open-cell polyurethane foam surface eventually employed was selected because it had been shown to be nontoxic and to induce a minimal inflammatory response.[5, 6] It was also considered that its structure would allow tissue ingrowth and eventual formation of an endothelial-like surface. The material has the additional advantage that it can be molded easily without special equipment.

In each of the patients in whom the valve was inserted a small diastolic pressure gradient was present afterward. This observation has suggested certain changes in the design of the valve which should further improve its hemodynamic function. Preliminary studies indicate that a prosthesis molded with the leaflets in a more closed position may be preferable. This modification, which results in a relatively larger anterior or aortic leaflet, also permits a larger orifice to be made without regurgitation. In the surviving patient the left atrial pressures recorded before and after operation do not necessarily reflect the degree of hemodynamic improvement. Before operation an atrial septal defect was present; this communication served to decompress the left atrium and had it not been present the left atrial pressure would undoubtedly have been higher preoperatively.

SUMMARY

Prosthetic mitral valves, designed to simulate the normal valve, have been constructed of an open-cell polyurethane foam reinforced with Dacron fabric. The leaflets are controlled by artificial chordae tendineae which are brought through the wall of the left ventricle, the length of the chordae can be adjusted to provide optimum competency of the valve. After experimental evaluation the prosthesis was employed for complete replacement of the mitral valve in 2 patients, 1 of whom survived. These patients are described and the details of the design and fabrication of the valve and the technique of insertion are presented.

ADDENDUM

May 23, 1960.—The surviving patient remains well following discharge from the hospital. In the fifth postoperative week she developed fever but repeated blood cultures showed no growth and the fever subsided promptly after the administration of penicillin and streptomycin. At this time a faint apical systolic murmur is audible.

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PARTIAL REPLACEMENT OF THE MITRAL VALVE WITH SYNTHETIC FABRIC

Harold King, M.D., Chien Sheng Su, M.D., and Joe G. Jontz, M.D., Indianapolis, Ind.

INTRODUCTION

As the technique of the open approach for visual correction of mitral valve disease becomes better developed, it is evident that direct plastic reconstruction of the valve leaflet would often be desirable. In many instances the use of foreign tissues or materials would be necessary to restore the valve to a more normal anatomic state. The easy availability of synthetic fabrics would make their use desirable in this location. Whether healing would occur and good function result when plastic cloth was substituted for part of an actively moving valve, subjected to high pressure changes, was not known. The present study was therefore undertaken.

METHODS

Mongrel dogs weighing 11 to 18 Kg. were used. Anesthesia was induced and maintained with intravenous methohexital sodium.* Respiration was controlled by insufflation of 100 per cent oxygen through an endotracheal tube. The animals were cooled to 33° C. in an ice bath. After they were removed from the bath, the esophageal temperature usually stabilized around 30° C. (28° to 32° C.).

A right thoracotomy was carried out through the fourth intercostal space. A large plastic tube was inserted into the right atrium for the gravity drainage of venous blood to the pump-oxygenator. The left femoral artery was cannulated for arterial inflow from the pump and the right femoral artery for monitoring of arterial blood pressure with a mercury manometer. A Sigmamotor pump and a large bubble oxygenator were used. The pump-oxygenator was primed with blood taken from donor dogs anesthetized lightly with methohexital sodium. Total cardiac bypass was established, usually at a flow rate of 40 c.c. per kilogram per minute. The heart was arrested with acetylcholine which was injected into the proximal aorta at a dosage of 10 mg. per kilogram of body

From the Department of Surgery, Indiana University School of Medicine, Indianapolis, Ind.

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*Furnished by the Lilly Laboratory for Clinical Research, Indianapolis, Ind.

weight. The left atrium was entered through the interatrial groove. The atrium was completely cleared of blood by an aspirator. In one group of animals, the central and basal portion of the aortic leaflet of the mitral valve was then incised close to and essentially parallel to the annulus. An elliptical piece of tissue, about 1 by 0.5 cm. in size, was excised. The defect was then grafted with a slightly larger piece of nylon or Teflon cloth. This was sutured into place with two or three running sutures of 5-0 arterial silk (Fig. 1, *a*).

In another group, the septal leaflet and attached chordae tendineae were excised but for a small rim of leaflet tissue next to the annulus. An artificial leaflet was then fashioned from synthetic fabric. The outline was similar to that of the normal leaflet. It was sutured above to the rim of leaflet tissue adjacent to the annulus, and below two extensions were sutured to each papillary muscle (Fig. 2, *a*). The left atrium and ventricle were then filled with saline solution and the left atrial wall was closed. The aspiration of the proximal aorta was carried out with a small needle before the aortic occlusive clamp was removed. In many instances, air was present in this location. If ventricular fibrillation occurred, it was converted to a more normal rhythm with electrical shock.

Total cardiopulmonary bypass time ranged from 30 to 50 minutes and cardiac arrest from 20 to 40 minutes. The animals were re-warmed by immersion in hot water at 42° C. until the esophageal temperature reached 35° C.

EXPERIMENTAL RESULTS

Initially, operations were done without sterile precautions. Many animals did not survive because of errors in surgical technique or in the management of the cardiopulmonary bypass. Air embolism was a particularly difficult problem. Some survived the initial postoperative period only to die from 3 to 7 days afterward from partial or total dehiscence of the suture line of the graft. Some failures resulted from infection and septic thrombosis of the left atrium; other failures were a result of suturing the plastic material to an area of the leaflet which was too thin and delicate. Dehiscences of the suture line were characteristically located near the free edge of the leaflet where the valve substance was thinnest (Fig. 1, *b*). These animals would characteristically do well for 1 to 3 days after the operation; they would then suddenly become ill and die within a few hours.

As a result of these experiences, the operative technique was altered so that the incision in the leaflet was located in the thicker, more basal portion close to the annulus. Separation of the suture line did not then occur. Five animals, operated upon under aseptic conditions, tolerated partial replacement of the major leaflet well and lived an active kennel life until the time of sacrifice. No cardiac murmurs were heard. In 3 of the 5, the left atrial and left ventricular pressures were taken 3 months after the grafting procedure. The pressure tracings were entirely normal and showed no evidence of mitral insufficiency. In 4, nylon cloth was interpolated into the valvular defect, and in the fifth, Teflon was used. The animals were sacrificed 3 months after the operative procedure. All of the grafts were invested with a layer of fibrous tissue which had

Fig. 1.—A group of illustrations showing the major leaflet of the mitral valve partially replaced with a graft of synthetic fabric.

a, A drawing which shows the usual position and form of the graft.

b, A photograph which shows partial dehiscence of the medial part of the suture line. The sutures remain attached to the graft. This animal made an excellent recovery until the third day after operation when it suddenly became ill and died from mitral regurgitation.

c, A photograph of the major mitral leaflet of an animal which was sacrificed 3 months after operation. The graft was made of nylon. The arrow points to a raised area near the center of the graft which was composed of an overgrowth of fibrous tissue.

d, A photograph of the major mitral leaflet of an animal which was sacrificed 3 months after operation. The arrow points to the graft. It was made of Teflon. The fabric is covered with a thin smooth layer of fibrous tissue.

a smooth endothelial-like surface. One showed a moderate amount of fibrous thickening in the region of the suture line (Fig. 1, *c*). In the others, the plastic cloth was covered with a very thin layer of fibrous tissue (Fig. 1, *d*). None demonstrated any significant contracture of the leaflet tissue itself.

Complete replacement of the major leaflet was carried out on 5 animals with aseptic precautions. Nylon cloth was used in 2. One lived 10 and the other 13 days. Both died in congestive heart failure. Autopsy revealed mitral

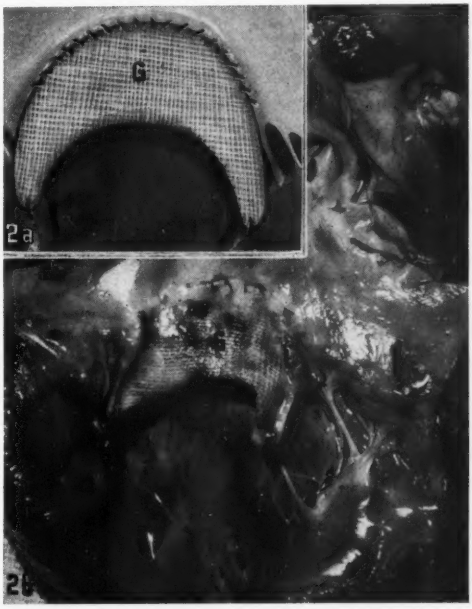

Fig. 2.—Two illustrations showing complete replacement of the major leaflet and attached chordae tendineae with synthetic fabric.

a, A drawing which shows the size and configuration of the artificial leaflet. The two lateral extensions of the graft were sutured to the papillary muscles. The extensions prevented eversion of the graft.

b, A photograph of an artificial leaflet of Teflon fabric. Following placement of the graft the animal remained in good health for 3 days and then became sick and died. Overwhelming mitral insufficiency resulted when a lateral extension of the graft became detached from a papillary muscle. The suture remained attached to the graft. The fabric is partially covered with a smooth layer of fibrin.

insufficiency to be present at both extremities of the graft. It was our impression that the valve function could be improved by increasing the area of the synthetic leaflet. Teflon cloth was used in the next 3 dogs. The grafts were made larger in size. One animal survived 18 days. Death resulted from dehiscence of the thoracotomy incision. Autopsy showed the valve to be competent. Another did well for 5 days and then died suddenly. Autopsy revealed one limb of the graft had become detached from the papillary muscle. Severe overwhelming mitral insufficiency resulted (Fig. 2, *b*). The third dog did well for about 6 weeks when it became sick and inactive. The animal was sacrificed at 7 weeks. Once again the graft was found to be separated from one of the papillary muscles.

Neither of the synthetic fabrics used in this experiment was entirely suitable for valve replacement. Sutures frequently pulled out of the nylon cloth even though the edge was carefully coagulated with heat. The nylon was thin and approximated the consistency of the leaflet tissue; however, it was inelastic and had a tendency to wrinkle. Sutures held well in the woven Teflon without special treatment of the cut edge. Unfortunately, the Teflon fabric which was commercially available was considerably greater in thickness and much less flexible than the mitral leaflet of the dog. Both fabrics were difficult to work with because of their inelasticity and relative inflexibility. Theoretically, the changing size of the mitral annulus and the normal variation in curvature of the leaflet from concave to convex with each heartbeat would make permanent pliability of the graft material most desirable. For replacement of a large area or a complete leaflet, elastic fabrics and living homologous tissues, such as atrial wall, should be investigated. The commercially available woven Teflon fabric should be satisfactory in most instances for partial leaflet replacement in patients.

Our results demonstrated that synthetic fabrics could be used successfully for partial replacement of the major leaflet of the mitral valve. The diaphanous character of the mitral leaflet of the dog made the technical problem of suturing the graft to the leaflet a difficult one. Good healing could not be expected if a portion of the suture line was located in the extremely thin medial area. When the graft was placed in the thicker basal part of the valve, better results were obtained.

Attempts to replace completely the major leaflet of the mitral valve were all eventually unsuccessful. The synthetic leaflets were made similar in configuration but slightly larger than normal. These functioned astonishingly well for short periods of time. Dehiscence of the attachment of an extension of the graft to a papillary muscle was the most common cause of failure. This fault undoubtedly could be corrected by a change in operative technique. Fabrics more suitable for valve replacement than those used in this study should also improve the results. Whether the flexibility of the graft would ultimately be lost as the fabric became covered with fibrous tissue was a question this study did not resolve. In the 1 animal that survived 7 weeks following complete replacement of the major leaflet, the fabric was relatively stiff at the time of autopsy. In view of the excellent functional results obtained in the early postoperative period, it would appear reasonable that complete leaflet replacement would be feasible once suitable replacement fabrics or tissues are found.

SUMMARY

1. Synthetic fabrics can be used successfully for partial replacement of the major leaflet of the mitral valve.

2. The problem of complete leaflet replacement is a difficult one and should be studied further. Some animals had good function of the mitral valve for short periods of time. All, however, eventually died from mitral insufficiency.

THE EVALUATION OF MITRAL STENOSIS WITH SELECTIVE LEFT VENTRICULAR ANGIOCARDIOGRAPHY

Viking Olov Björk, M.D., and Herman Lodin, M.D., Uppsala, Sweden

LEFT ventricular angiocardiography is never primarily indicated for the evaluation of the stenotic component in mitral valvular disease. Introduction of contrast medium direct into the left atrium by percutaneous injection over the ninth rib paravertebrally on the right side will provide much more reliable information about a mitral stenosis when this is needed. In the course of left ventricular angiocardiography in the evaluation of mitral insufficiency and aortic stenosis we have observed a typical doming of the stenosed mitral valve. After correlation of this roentgenologic sign with the findings at surgery, we consider it to be of significant diagnostic value.

METHOD

The patient is examined in the supine position, a needle being introduced through the apex beat and directed toward the second right costochondral junction. The procedure is performed under local anesthesia. One to one and one-half milliliters of 76 per cent Urografin per kilogram of body weight is injected at a rate of 30 ml. per second. The exposures (maximum time, 0.03 sec.) are made in two planes (frontal and lateral projection) with a frequency of 6 frames per second during and 1 to 2 seconds after the injection. The examination is concluded with a series of frames taken at a frequency of 1 to 2 per second. During and 1 to 2 seconds after the injection, the carotid arteries are compressed in order to reduce the flow of contrast medium to the brain.

RESULTS

1. *Open Mitral Orifice.*—No dome formation was found in cases in which there was no mitral stenosis. One patient with a normal heart was investigated (Fig 1, *A* and *B*). In this case the blood from the left atrium during diastole rapidly found its way into the left ventricle causing a filling defect in the valvular area. In the anterior projection, however, the defect was sharply outlined in its inferior part only, and not all around, as will be demonstrated in the cases with typical doming of the fused mitral valves. In most patients with no mitral stenosis, the anterior projection will show at most a poorly defined defect in the ventricular contrast (Fig. 2, *A*).

From the University Hospital, Uppsala, Sweden.
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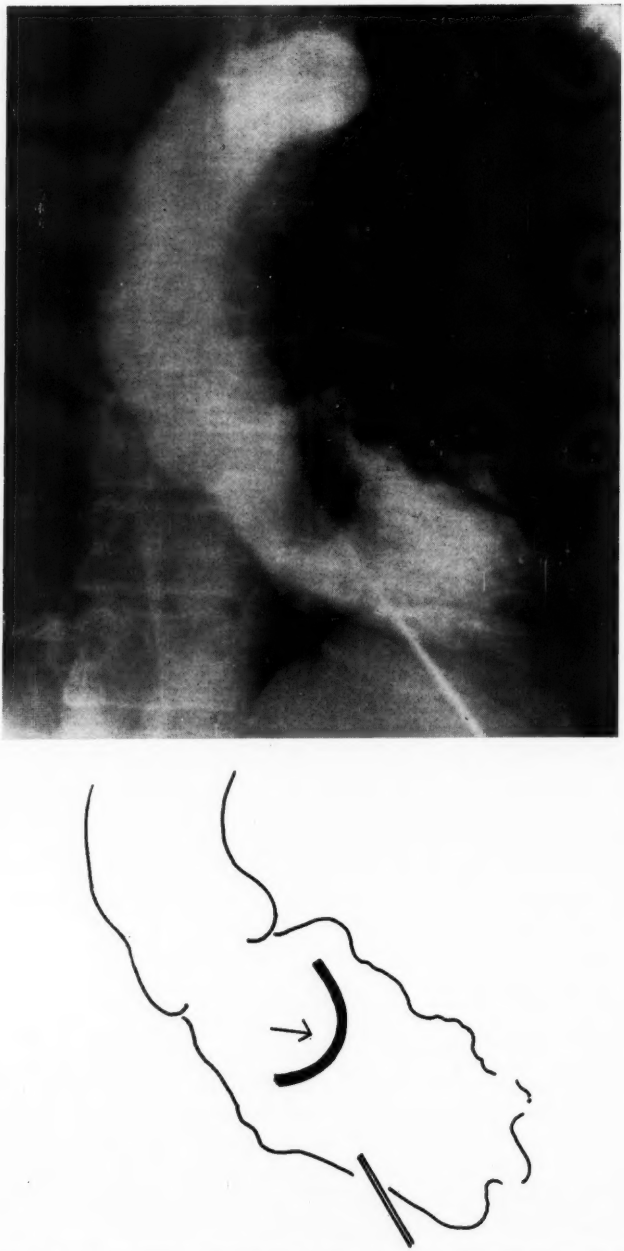


Fig. 1.—A 42-year-old man with a normal heart and a malignant tumor of the thymus. Left ventricular angiocardiograms in ventricular diastole. *A*, Frontal projection. In the upper part of the left ventricle a filling defect the size of a walnut, with a sharp inferior border (→).

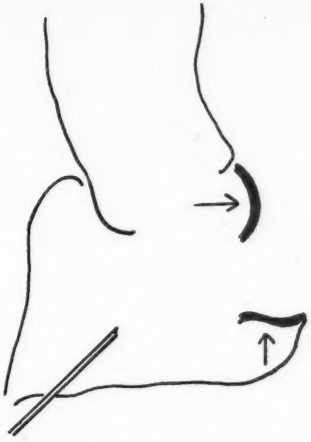


Fig. 1.—Cont'd. *B*, Lateral projection. The mitral leaflets are partly visualized. The posterior leaflet (↑) is more or less parallel to the x-ray beam and is therefore responsible for the sharp inferior border of the defect in the anterior projection. The anterior leaflet (→), however, is oblique to the x-ray beam and is not distinctly outlined in the anterior projection. The area between the leaflets has a poorly defined anterior border, caused by the rapid mixing of the contrast medium with blood from the left atrium, No doming.

In cases of a patent mitral orifice, the leaflets may be seen in the lateral projection during diastole (Figs. 1, *B* and 2, *B*). The valvular area is not sharply outlined anteriorly, owing to the rapid emptying of the left atrium and immediate dilution of the contrast medium in the left ventricle; thus, no dome is visible.

2. *Mitral Stenosis.*—When the mitral valves are fused to form a dome during diastole, this dome will bulge into the left ventricle making a sharply outlined filling defect in the left ventricle. This formation is seen in both the

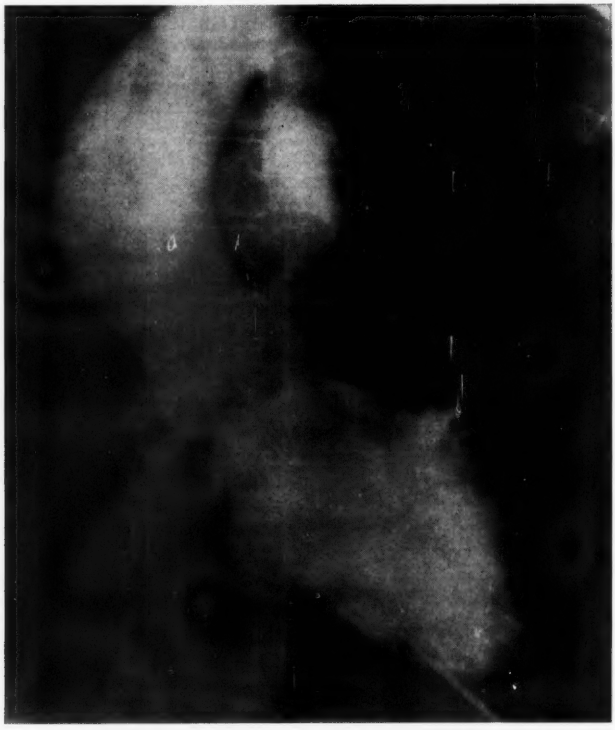


Fig. 2.—A 27-year-old man with pure aortic stenosis. Left ventricular pressure was 164/20 mm. Hg. Pressure gradient over the aortic valves was 27 mm. Hg. Pulmonary capillary pressure was 9 mm. Hg. Pulmonary artery pressure was 20 mm. Hg. Left ventricular angiocardiograms in ventricular diastole. *A*, Frontal projection. In the mitral valvular area of the left ventricle a small, poorly defined, filling defect can be seen, caused by the inflow of blood from the left atrium.

frontal and lateral projections (Figs. 3, *A* and *B*, and 4, *A* and *B*). In all 20 patients operated upon, in which a dome had been found preoperatively in the left ventricular angiocardiogram, mitral stenosis was found at operation. The stenosis was severe in all but 1 case in which it was very mild, admitting 1½ fingers.

In cases of combined mitral stenosis and mitral insufficiency, it may be difficult to demonstrate doming if the insufficiency is severe, since contrast medium then becomes mixed with blood in the left atrium and no distinct

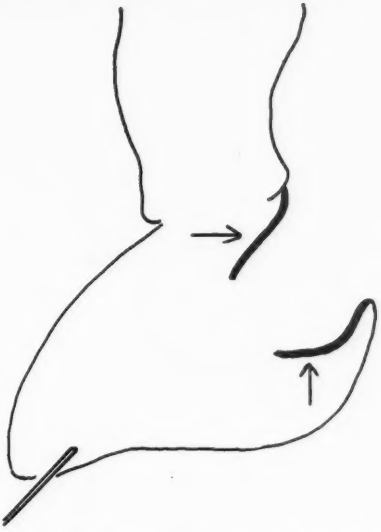


Fig. 2.—Cont'd. *B*, Lateral projection. The open mitral orifice is seen and the mitral leaflets are partly visualized. Rapid mixing of the blood from the left atrium and the contrast medium between the leaflets causes an anterior border that is not sharp. No doming. Anterior (→) and posterior (↑) mitral leaflet.

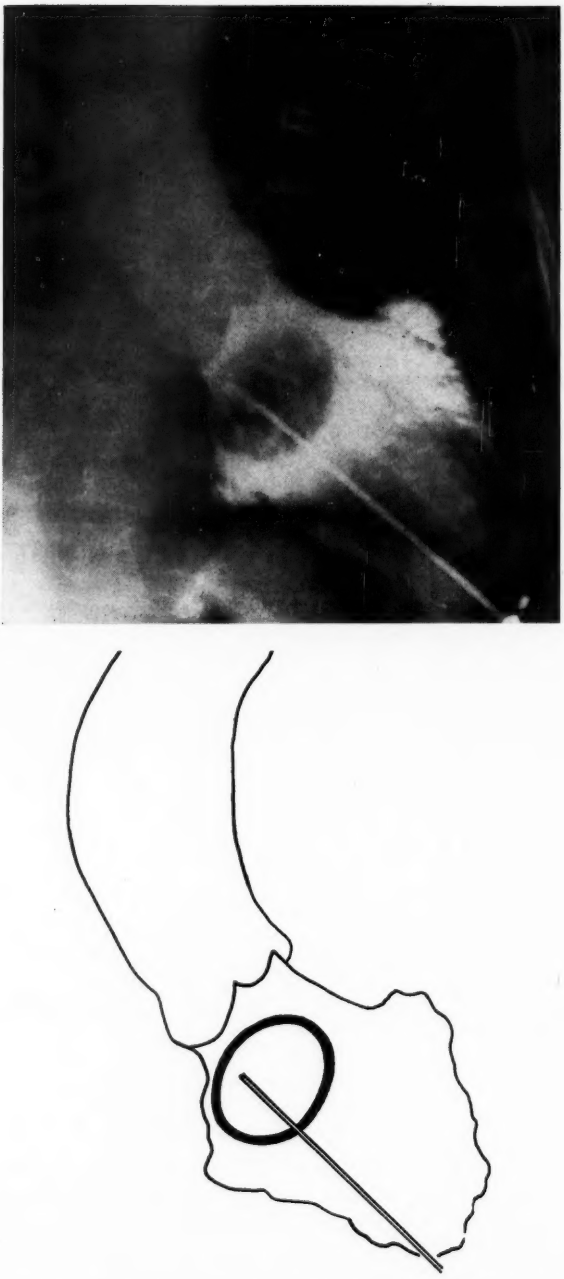


Fig. 3.—A 49-year-old woman with mitral and aortic stenosis (no pressure gradient over the aortic valves) and mitral insufficiency. At operation there was a rigid mitral stenosis with an opening, 10 mm. long. Transventricular dilatation of both mitral and aortic stenosis. Left ventricular angiocardiograms in ventricular diastole. *A*, Frontal projection. Inferior to the aortic orifice an oval, clearly outlined, ventricular filling defect is seen. The defect is caused by the fused mitral leaflets. True mitral dome formation.

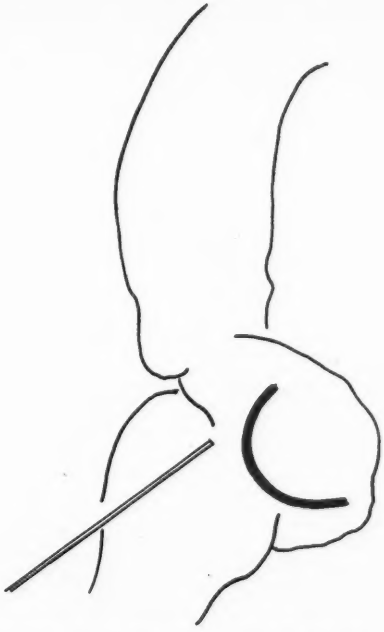


Fig. 3.—Cont'd. *B*, Lateral projection. The fused mitral leaflets form a sharply outlined dome bulging into the ventricle.

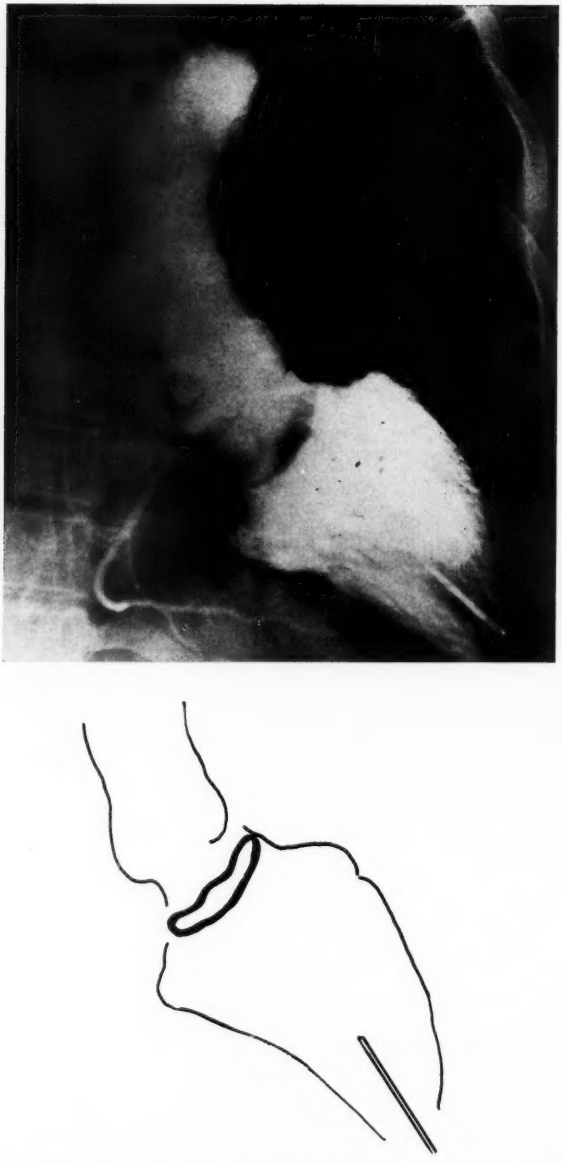


Fig. 4.—A 42-year-old woman with mitral re-stenosis. At reoperation, the mitral orifice was found to admit the tip of the little finger. Pulmonary artery pressure was 15 mm. Hg. pulmonary capillary pressure (systolic) was 30 mm. Hg—during work it was 57 mm. Hg. Left ventricular angiocardiograms in diastole. *A*, Frontal projection. Slit-like, sharply contoured defect in the mitral valvular area. In this case the inferior border is caused by the anterior leaflet and the superior border of the posterior leaflet.

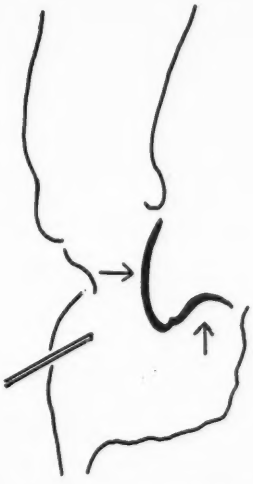


Fig. 4.—Cont'd. *B*, Lateral projection. The anterior leaflet (→) bulges forward, and its border is pulled down. The posterior leaflet (↑) is thickened, and its upper border is projected above the border of the anterior leaflet.

difference between atrial and ventricular contrast density can be made out. In these cases the dome will only be outlined at the beginning or the end of the cycle.

3. *The False Dome.*—It is most important to see the doming of the mitral valves in both anterior and lateral views to establish the diagnosis of mitral stenosis. In the lateral projection the atrial wall may simulate a dome caused by fused mitral valves, but in such cases no dome can be seen in the antero-posterior projection. Furthermore, this false dome in the lateral projection will persist during systole (Fig. 5, *A-D*).

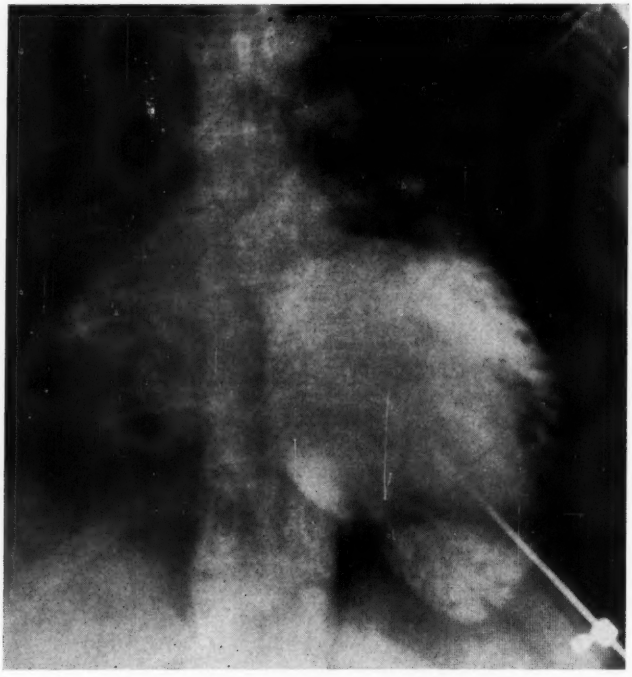
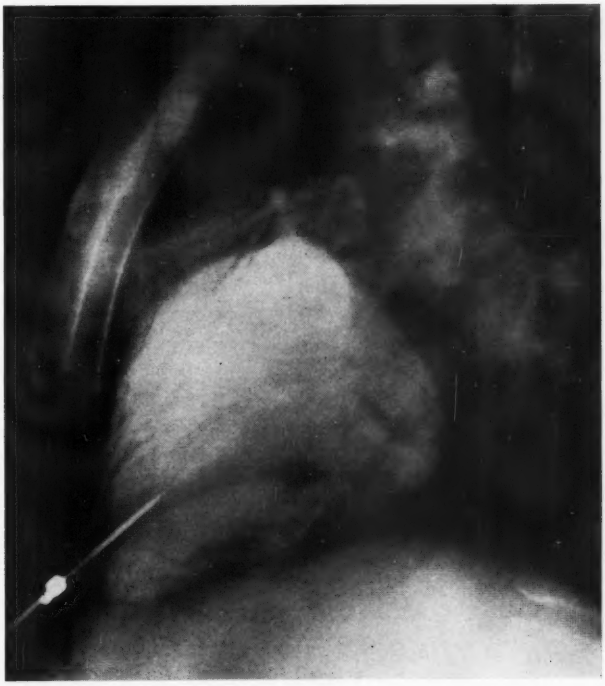


Fig. 5.—A 31-year-old woman with pure mitral insufficiency. Exploratory cardiotomy revealed a severely incompetent mitral orifice admitting 2 fingers. Left ventricular angiocardiograms. *A, Frontal projection.* Diastole. No ventricular filling defect.

CONCLUSION

Ordinarily the inferior border of the filling defect seen in the anterior projection may be sharply outlined and caused by the mitral annulus itself, the short posterior mitral leaflet lying tangential to the x-ray beams in diastole (Fig. 1). Usually the superior border is not distinctly outlined since, in diastole, the anterior mitral leaflet is projected obliquely to the plane. Furthermore, the rapid emptying causes immediate dilution of the contrast medium resulting in a poorly defined border between this and blood. A diagnosis of mitral stenosis can only be accepted if, (1) the contrast defect is observed in both planes, and (2) the defect in the anterior plane is sharply outlined all



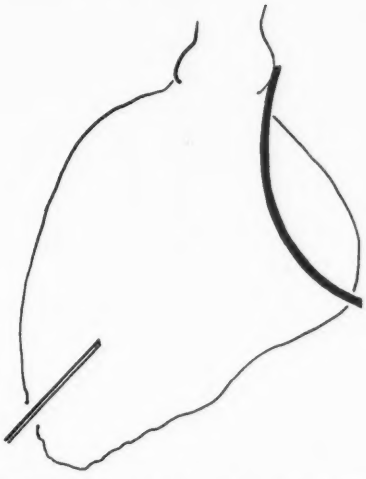


Fig. 5.—Cont'd. *B, Lateral projection.* Diastole. Dome formation resembling true mitral doming.

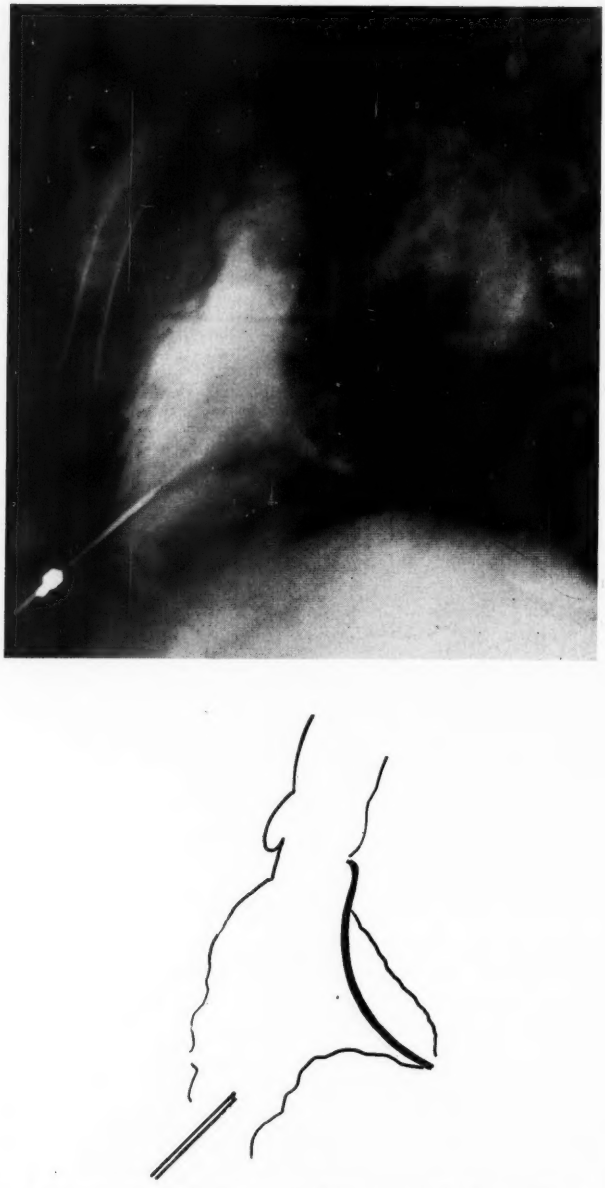


Fig. 5.—Cont'd. *C*, Systole. The dome formation is unchanged.

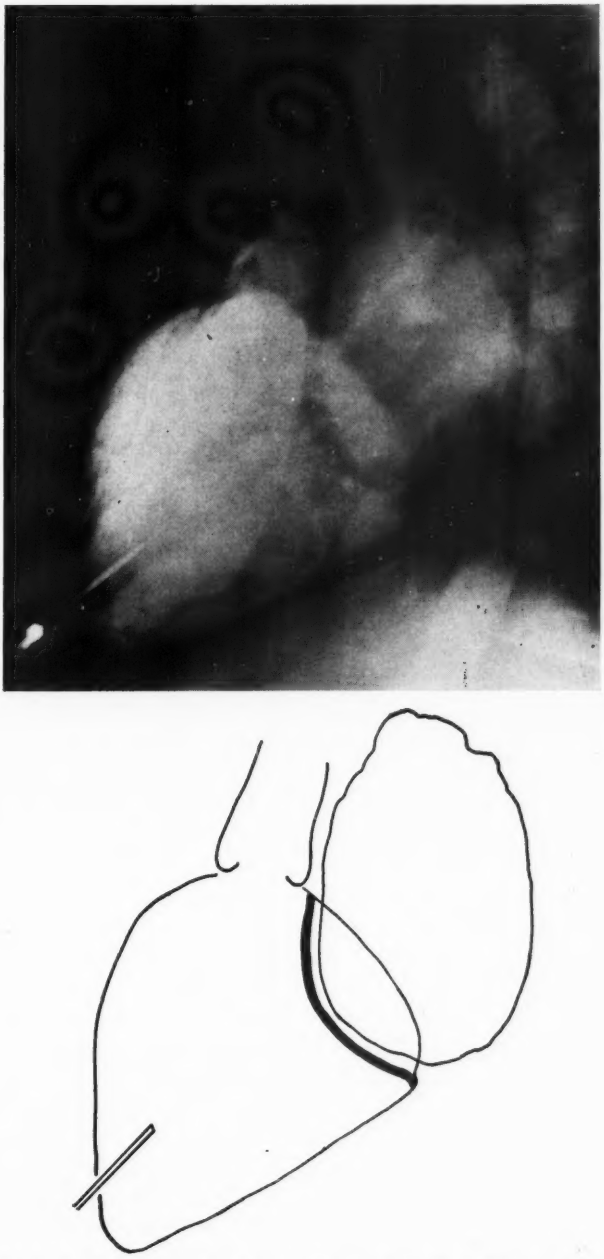


Fig. 5.—Cont'd. *D*, Diastole. The left atrium filled with contrast medium. The dome formation corresponds to the atrial wall.

around. This is due to the slow emptying of blood from the left atrium and the forward bulging of the fused mitral valves. The doming will cause one part of the anterior leaflet to lie parallel to the x-ray beams and therefore to be visualized. Thus, the anterior mitral leaflet will act as a bulging sail behind which a pool of contrast-free blood projects into the left ventricle (Fig. 6).

In most cases the filling defect has been rounded, but owing to the relation between an eccentric orifice and the projection used, the defect may appear more slit-like and the border of the anterior leaflet may sometimes even be viewed below that of the posterior leaflet (Fig. 4).

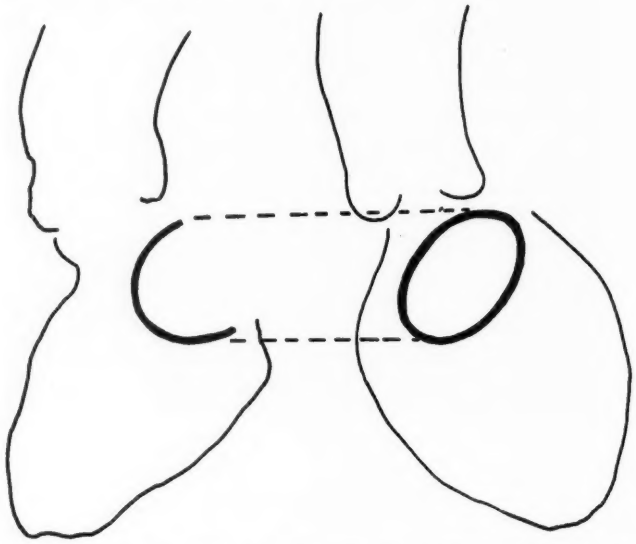


Fig. 6.—Diagram which illustrates the visualization of the mitral dome. *Left side*—Lateral projection. *Right side*—Frontal projection.

The absence of a dome in the anterior projection in cases of an enlarged left ventricle cannot exclude the presence of a mitral stenosis, as excessive contrast medium in the left ventricle will mask the dome.

SUMMARY

During a series of selective left ventricular angiograms, we have observed a clearly visible and sharply outlined filling defect in the left ventricle caused by the fused mitral valves forming a dome and projecting down into the left ventricle. When this dome is visible, sharply outlined in both lateral and anteroposterior projections, it constitutes evidence for the diagnosis of mitral stenosis. It must be distinguished from the left atrial wall, which will be seen projecting into the left ventricle only in the lateral projection.

The roentgenologic diagnosis has been confirmed at operation.

TRICUSPID ATRESIA

Treatment by Superior Vena Cava–Pulmonary Artery Anastomosis

Gordon R. Cumming, M.D., F.R.C.P.(C), Colin C. Ferguson, M.D., F.R.C.S.(C), James N. Briggs, M.D., M.R.C.P., and Edward G. Brownell, M.D., F.R.C.P.(C), Winnipeg, Manitoba

AFTER much animal experimentation,[1-4] Glenn reported the clinical application of a superior vena cava to pulmonary artery anastomosis. This unique anastomosis has had a longer and more extensive trial in Russia[5] and in some centers is favored over the Blalock or Potts types of anastomosis for cyanotic congenital heart disease. Tricuspid atresia is one anomaly not yet amenable to open-heart surgery and in the following case such gratifying results were obtained by a superior vena cava to pulmonary artery anastomosis that it would seem that further attention should be drawn to this procedure.

CASE REPORT

T. H., a girl, was born 3 weeks prematurely. Pregnancy, delivery, and neonatal course were uneventful. A cardiac murmur was noted at age 3 days. At the age of 6 weeks, the infant was doing well, no cyanosis was noted, but the murmur was still present. At 7 weeks of age, following a mild gastrointestinal upset, cyanosis developed suddenly, and the infant was in severe distress. On admission to the hospital, cyanosis was severe, pulse was 200, liver edge was 3 cm. below the costal margin, and a Grade 3 systolic murmur was heard along the left sternal border. Severe cyanosis persisted despite oxygen therapy and digitalis, and two anoxic attacks were observed.

X-ray studies of the chest showed moderate cardiac enlargement, a concave pulmonary artery segment, decreased lung vascularity, and a pneumonic area in the right middle lobe. The electrocardiogram showed left axis deviation (minus 30 degrees) and a clinical diagnosis of tricuspid atresia was made. An angiocardiogram, with a catheter high in the inferior vena cava, showed (Fig. 1) the atresia of the tricuspid valve and a small patent foramen ovale. Additional films (Fig. 2) showed this to be a type 1b of Keith and associates[6] with a small ventricular septal defect, a rudimentary right ventricle, and a small pulmonary artery. The aortic arch was on the right side.

Using the operative technique of Glenn,[4] the right pulmonary artery was anastomosed to the superior vena cava and the junction of the latter to the right atrium was occluded (Fig. 3). The entire operative procedure lasted less than one hour.

From the Departments of Medicine and Surgery of the Children's Hospital, and of the University of Manitoba, Winnipeg, Manitoba, Canada.

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Fig. 1.

Fig. 2.

Fig. 1.—An angiocardiogram, posteroanterior view, which shows atresia at the tricuspid valve area, and a jet of medium traversing a patent foramen ovale.

Fig. 2.—Later film, lateral view, which shows the rudimentary right ventricle and small pulmonary artery.

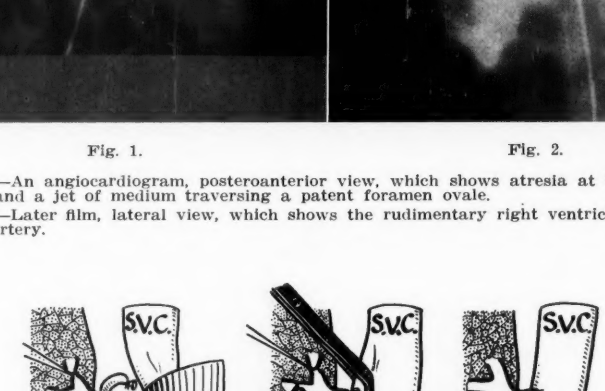

Fig. 3.—Operative procedure. *A*, Ligation of the proximal end of the right pulmonary artery. *B*, End-to-side anastomosis of the pulmonary artery to the superior vena cava. *C*, Ligation at the superior vena cava right atrial junction.

An immediate postoperative improvement in the cyanosis was noted, although for the first 4 postoperative days moderate puffiness and cyanosis of the face, neck, and arms was noted. The infant was discharged 12 days after operation. When seen at age 5 months, 14 weeks after surgery, the infant continued to do well. The weight was 5.6 kilograms compared to a birthweight of 2.5 kilograms and a weight at 7 weeks of 3.3 kilograms. Heart size showed little change. Auscultatory findings were unchanged and there was no continuous murmur. There was no facial puffiness, although venous pressure in the right arm was 19 mm. Hg. The ear oximeter showed 85 per cent oxygen saturation, bearing out the just detectable clinical cyanosis. Injection of opaque medium into a right antecubital vein showed the anastomatic area and the filling of the right pulmonary artery from the superior vena cava.

DISCUSSION

Early surgery is necessary in tricuspid atresia. Half of all cases fail to survive past 6 months of age.[6] The type reported rarely survives infancy.[7] Pulmonary artery thromboses may occur and preclude successful surgery at a later date.[8] Thus, any surgical procedure for tricuspid atresia must be suitable for the small infant. It would appear that a superior vena cava to pulmonary artery anastomosis is not only suitable for small infants, but is technically a more simple procedure and also has theoretical physiologic advantages over the Blalock and Potts anastomoses in cases of tricuspid atresia.

In a series reported by Neill,[9] only 30 per cent of patients, under one year of age, with tricuspid atresia survived a Blalock procedure. In many cases, while anoxia was corrected, left ventricular failure occurred. In the common type of tricuspid atresia the left ventricle is doing all the work of the heart, and standard aortic to pulmonary artery anastomoses further increase the load on the left ventricle. On the other hand an anastomosis of the superior vena cava to the pulmonary artery corrects anoxia without necessarily increasing the load on the left heart. Glenn and co-workers[3] showed that an anastomosis of the inferior vena cava to the pulmonary artery leads to ascites and chronic morbidity. In man further help may be obtained by full use of gravity to aid blood flow. The patient reported has been maintained in a 30 degree head-up position since surgery. The future course is unknown. Growth of anastomotic sites is a vexing problem but it applies to arterial as well as to venous anastomoses. Collateral venous channels may develop between the superior and inferior caval systems so that progressively less percentage of the blood from the upper part of the body will reach the right lung. Thromboses may occur, perhaps more often than with arterial anastomoses. However, Bakulev and Kolesnikov[5] claimed better long-term results with vena cava–pulmonary artery anastomoses than with Blalock or Potts procedures. In view of the advent of total corrective surgery by cardiopulmonary bypass techniques, this type of anastomosis is probably best reserved for cases such as tricuspid atresia in which total anatomic correction is not yet possible.

SUMMARY

The anastomosis of the superior vena cava to the right pulmonary artery has had extensive laboratory application and is physiologically a good operation

which may be used to bypass the right heart in cases of tricuspid atresia. The operation increases pulmonary blood flow without increasing left ventricular work. Technically the operation is simple. A successful application of this procedure in a sick 7-week-old infant with tricuspid atresia is reported and discussed.

ADDENDUM

Since submitting this paper for publication, 3 additional patients with tricuspid atresia have had a superior vena cava to right pulmonary artery anastomosis. Two, aged 5 and 10 months of age, have done well, but in the third patient, aged 8 days, it was not possible to perform a successful anastomosis and death occurred 8 hours after surgery. The patient reported continues to do well.

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PRESSURE CONDITIONS IN THE PULMONARY CIRCULATION DURING CARDIAC BYPASS BY DOUBLE PUMPS

H. Aletras, M.D., Athens, Greece, J. Garibotti, M.D., Cordoba, Argentina, E. Kolb, M.D., Zurich, Switzerland, M. Noirclerc, M.D., Marseille, France, J. Osorio, M.D., Mexico City, Mexico, W. Tomaszewsky, M.D., Warsaw, Poland, G. William-Olsson, M.D., and Å. Senning, M.D., Stockholm, Sweden

IN SEARCHING for reliable methods of cardiac bypass for open cardiac surgery, several techniques for the oxygenation of blood have been tried. Isolated homologous and heterologous lungs were used (Mustard[8, 9]) as well as autogenous lungs (Wesolowsky and Welch,[14] Read and co-workers,[12] Blanco, Schaefer, and Bailey,[1] Drew,[5, 6] Shields and Lewis,[13] Mustard, Sapirstein, and Pav,[10] Helmsworth and associates[7]). The use of the autogenous lungs for oxygenation has been technically rather complicated, especially in cardiac malformations such as septal defects. The interest for this method diminished when promising artificial oxygenators were developed. However, the artificial oxygenators have their limitations and, with long periods of extracorporeal circulation, the mortality is high. There seems experimentally to be a decrease in the diffusion capacity of the lungs (Pitzele and Senning[11]) which is a serious problem especially in patients with low pulmonary reserve, for example, with ventricular septal defect combined with pulmonary hypertension. The cause of this decrease in pulmonary diffusion capacity is not known but may be due to the fact that the lung is the only organ that is not perfused during the period of bypass. The trend has been therefore to find a suitable technique of using the autogenous lung in cardiac bypass.

A right-sided bypass can be suspected of having an unfavorable effect on the pulmonary vascular system as pulmonary edema often appears after bypass (Blanco and associates[2]). These experimental series have been made to study changes in pressures in the pulmonary circulation during cardiac bypass by an automatic double pump, using gravity drainage and the autogenous lung for blood oxygenation.

TECHNIQUE

Adult mongrel dogs, weighing 13 to 29 kilograms, were anesthetized with intravenous Pentothal intubated, and ventilated with an Engström respirator.

From the Surgical Research Laboratory, Thoracic Clinic, Karolinska Hospital, Stockholm, Sweden.

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The femoral arterial pressure was recorded. Thoracotomy was made through the fourth intercostal space.

Bypass was performed with a modification of the pump used in the Crafoord-Senning heart-lung machine* (Crafoord, Norberg, and Senning[3]) (Fig. 1). Eleven experiments were performed at normal body temperature. In nine experiments, the body temperature was lowered to about 20° C. with a disposable

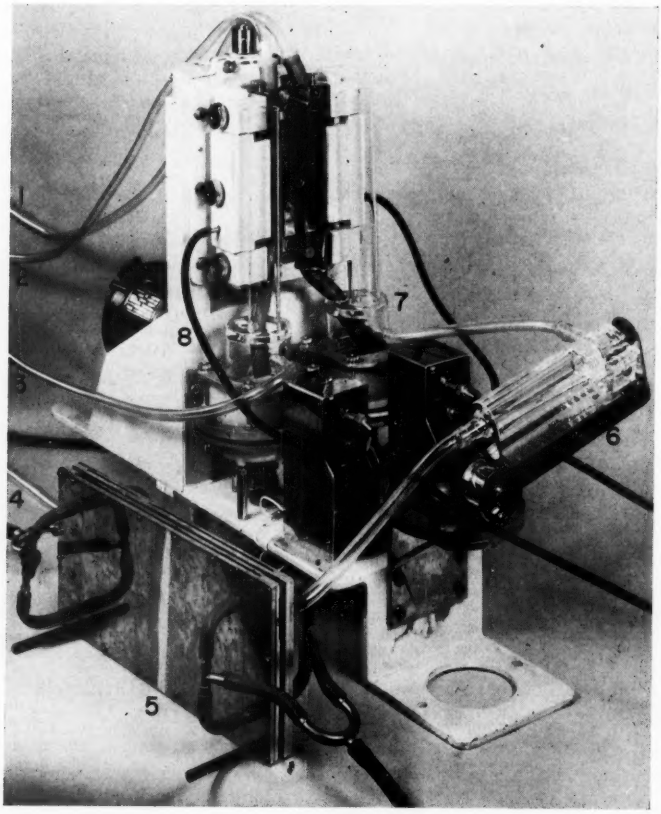

Fig. 1.—Double pump for cardiac bypass. *1*, Systemic arterial line. *2*, Pulmonary arterial line. *3*, Systemic venous line. *4*, Pulmonary venous line. *5*, Heat exchanger. *6*, Flow meter. *7*, "Left heart" regulation vessel. *8*, "Right heart" regulation vessel.

heat exchanger in the "venous" side of the left heart pump. This heat exchanger consists of a thin-walled (15 by 30 cm.) plastic bag placed between two metal plates cooled by tap water. The distance between the metal plates can be adjusted from 1 to 4 mm. to regulate the resistance. Heparinized blood (25 mg. per 400 c.c. blood) was used to fill the system and the dog received

*Manufactured by AGA, Lidingö, Sweden.

4 mg. per kilogram of body weight of heparin. Bypass was, in these experiments, started simultaneously in both circuits. Bypass of the right heart was made, draining the auricle by gravity to the reservoir of the pump through an intercalated flowmeter of rotameter type. The blood was pumped to the pulmonary artery by a cannula introduced through the outflow tract of the right ventricle. During bypass the pulmonary artery was strangulated round the inserted cannula. In four experiments, one pulmonary artery was occluded and perfusion done only to the other lung.

The left heart was bypassed in a similar way. Blood was drawn from the left atrium through a cannula via the heat exchanger into the reservoir of the arterial pump and pumped back to the systemic circulation through the subclavian artery. The pumps automatically pumped back all blood received from the atria. The flow was thus mainly dependent on the total circulating blood volume in the machine and the body. It was kept at a level where the

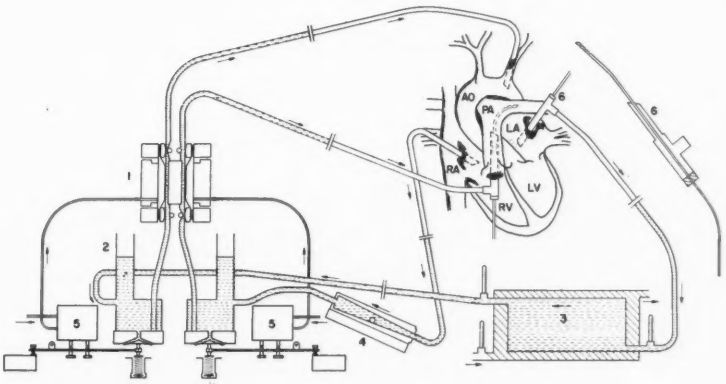

Fig. 2.—Schematic illustration of cardiac bypass by double pump. *1*, Double pump. *2*, "Left heart" regulation vessel. *3*, Heat exchanger. *4*, Flow meter. *5*, Pump cuff-pressure regulating valve. *6*, Left auricular cannula with inserted pressure-recording cannula.

blood pressure during bypass was about the same as before the perfusion, except in the nine experiments in which hypothermia was used. In six of these experiments, the flow was diminished during cooling from 50 to 30 ml. per kilogram of body weight per minute. Undesired variations in flow were corrected by adjustment of the blood volume.

Pressures in the pulmonary artery, left atrium and femoral artery were recorded with the reference point 8 cm. above the operating table by cannulae inserted in the pulmonary artery, the left atrium, and the femoral artery (Fig. 2).

The bypass lasted 60 minutes in eleven experiments at normothermia, and 80 minutes in eight hypothermia experiments, and 120 minutes in one. Cooling and rewarming periods were 20 to 35 minutes. After finishing bypass, protamine chloride was given until coagulation time was about 5 minutes; the blood volume was adjusted to the calculated pre-perfusion value.

RESULTS

Normothermia.—There were eleven bypass experiments under normothermia. In Fig. 3 the curves represent the average arterial, pulmonary artery, and left atrial pressures as well as the flow during bypass.

At an average flow rate of 50 ml. per kilogram of body weight per minute, the arterial pressure was maintained at the same level as before bypass. After perfusion was finished, a fall in arterial pressure was constant. It returned to normal when the blood volume was corrected; usually a blood transfusion of 100 to 200 ml. was given.

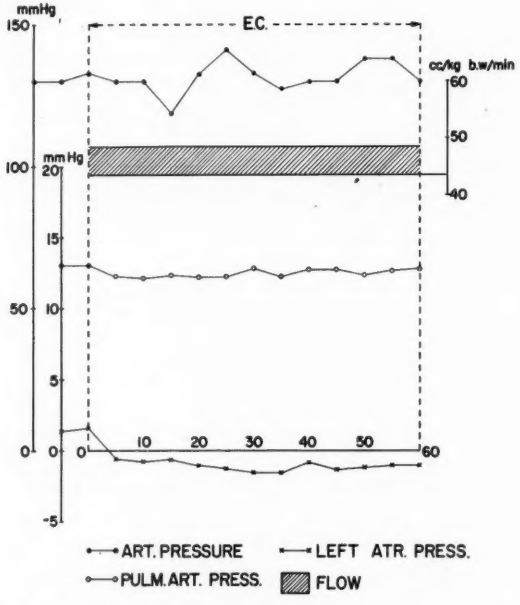

Fig. 3.—Average mean values of pressure in systemic artery, pulmonary artery, and left atrium during bypass with double pump in eleven experiments in which blood flow was kept constant and body temperature normal.

The pre-perfusion pressure in the pulmonary artery was about 13 mm. Hg. It remained stable with insignificant fluctuations throughout the experiments in all but 1 dog. This dog had a pulmonary artery pressure of 25 mm. Hg prior to the bypass, which rose to a maximum of 32 mm. Hg during bypass and fell to 27 mm. Hg at the end of it. In this dog, the flow was 80 ml. per kilogram of body weight per minute. In the case in which one pulmonary artery was clamped, the pressure in the main pulmonary artery did not increase significantly.

The pressures found in the left atrium averaged 1.5 to 2 mm. Hg before bypass and dropped to –0.5 mm. Hg when perfusion started and to –1 mm. Hg at the end of it. After perfusion was finished it immediately became normal.

There were no signs of pulmonary edema in any of the perfused normothermic dogs.

Hypothermia.—In nine experiments, extracorporeal circulation was combined with cooling. Fig. 4 shows the recordings of one typical experiment in which the flow was allowed to fall. In the experiment shown in Fig. 5, the flow was kept constant. Cooling was performed during the first 20 to 25 minutes of perfusion and rewarming during the last 25 to 30 minutes. With falling temperature, the pressures fell in the systemic arteries, the pulmonary artery, and the left auricle. In three experiments, however, there was a short rise in arterial and pulmonary artery pressure during the first minutes after the start of perfusion with cold blood, as is shown in Fig. 5.

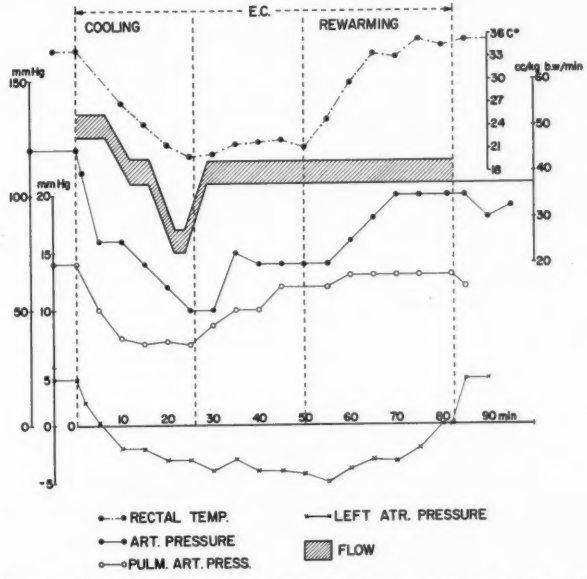

Fig. 4.—Mean values of pressure in systemic artery, pulmonary artery, and left atrium, blood flow and rectal temperature during bypass with double pump combined with internal cooling. A typical experiment in which the blood flow was allowed to fall.

During rewarming, the pressures rose and afterward showed the same pattern as in the experiments without hypothermia.

There were no signs of pulmonary edema in any of the perfused hypothermic dogs.

DISCUSSION

It seems possible that the high mortality after long periods of extracorporeal circulation is caused, in some cases, partly by a decreased pulmonary diffusion capacity due to the fact that the lung is not perfused during bypass, or partly because the capillaries are occluded by blood particles, or partly because of a general effect of blood trauma. These injurious factors can be avoided or

diminished by excluding the artificial oxygenator through using the autogenous lung as an oxygenator. The lungs are then perfused and the blood trauma from artificial oxygenation is avoided. In combination with cooling, the perfusion volume can be decreased and, as the circulation is supported by pumps,

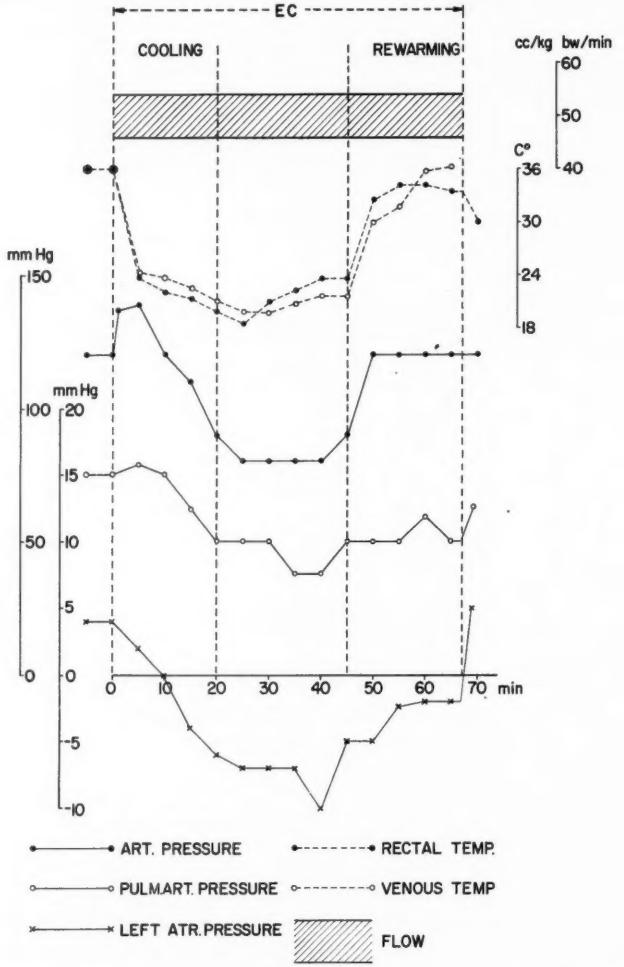

Fig. 5.—Mean values of pressure in systemic artery, pulmonary artery, and left atrium, as well as blood flow, rectal and venous temperatures during bypass with double pump combined with internal cooling. A typical experiment in which the blood flow was kept constant.

deep hypothermia can be applied. The flow can then be extremely small and periodically arrested, and thus the blood trauma also is diminished in the pumps. This technique seems to be of special importance in coronary surgery when long extracorporeal circulation periods are necessary.

This experimental series has been performed in order to see if pulmonary vascular changes occur during cardiac bypass performed by double pumps, with or without internal cooling.

In the series of eleven experiments at normothermia, there were no changes in the pulmonary artery pressure, if the starting pressure was normal and the flow was kept at a level where the blood pressure in the systemic arteries was the same as before bypass. As the collecting vessels of the pump were placed at a low level, the pressure in the left atrium fell below zero in the central part of the atrium. This minimized filling of the left ventricle from the atrium. After finished perfusion, the left atrial pressure immediately reverted to the pre-perfusion value. The pressure showed an insignificant rise during bypass at a flow of 80 ml. per kilogram of body weight in only 1 dog with slight pulmonary hypertension before bypass. This returned to normal, as in the other experiments, after perfusion was ended. No changes in the pulmonary vascular resistance during bypass were found in this series of eleven experiments.

In the other series of nine experiments, in which bypass with double pumps was combined with internal cooling to a body temperature of 20° C., the flow was kept constant in three experiments, and diminished during the period of cooling in six experiments. In all experiments, there was a decrease in the pulmonary artery pressure.

The pressure in the left atrium was recorded in the region of the mitral valves where the cannulae hinder the atrium from collapsing, thus making a negative pressure possible. This pressure is not, on the other hand, representative for the pulmonary veins. They collapse when the pressure is zero and the thorax is open. The increased pressure gradient between the pulmonary artery and the left atrium thus is an artefact only; there is no real increase in pulmonary vascular resistance. When the temperature of the experimental animal is normalized and the perfusion is ended, the pulmonary artery and left atrial pressures have reached their original pre-perfusion levels.

This experimental series has been part of our training program. Therefore the mortality has been high because of technical errors. When the most experienced among us performed the perfusion, 6 animals out of 8 survived after 1 hour of perfusion; and two out of three survived in experiments when there was 80 minutes of perfusion with the animals under hypothermia. The cause of death was postoperative bleeding and infection.

Experimentally there were no signs of pulmonary circulatory damage when this technique of cardiac bypass was applied. The method seems to have its application in clinical cardiac surgery.

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SYSTEMIC HYPERTENSION COMPLICATING THE LIGATION OF PATENT DUCTUS ARTERIOSUS

Frederick H. Taylor, M.D., Francis Robicsek, M.D., Paul W. Sanger, M.D., Robert E. Stam, M.D., and Terry T. Rees, M.D., Charlotte, N. C.

THE development of sclerotic changes in the pulmonary vessels and consequent pulmonary hypertension are common complications of patent ductus arteriosus. If these sclerotic changes are far advanced, the blood pressure in the pulmonary artery may approximate the pressure of the aorta and the original left-to-right shunt becomes "bi-directional" or even reversed. In cases of severe pulmonary hypertension, the ductus serves as a vent which decompresses the overloaded lesser circulation.[1, 2, 4, 5, 8, 9, 16, 17] The dangers of ligation of the ductus in such conditions were recognized early.[8-11, 18] The closure of this "emergency outlet" may lead to an immediate increase of the blood pressure in the pulmonary artery and cause an acute insufficiency of the right ventricle, which is usually fatal.

The pressure relations in the systemic circulation are less affected by the pressure of the ductus.[18] Because of the decrease of the total systemic circulatory resistance, the diastolic pressure is usually low; the systolic pressure remains normal.[14, 15] Following ligation of the ductus, the diastolic pressure returns to normal. Holman has described an increase also in the systemic systolic arterial pressure following operative closure of patent ductus arteriosus. He attributed this temporary rise in pressure to increased blood volume. The pressures in these cases returned to normal when the blood volume became normal.[7-9]

One of our patients, on whom a surgical closure of a patent ductus arteriosus was performed, experienced a very unusual complication, i.e., the development of an acute, severe, permanent systemic hypertension.

CASE REPORT

A 38-year-old white woman was admitted on May 18, 1957, to the Department of Thoracic Surgery, Mercy Hospital. She was known to have had heart disease since infancy. Her symptoms were those of exertional dyspnea, some pain in the mid-sternal area, palpitation, and nonproductive cough. These symptoms became more pronounced during the 4 months previous to admission.

From the Department of Thoracic Surgery and Heineman Cardio-Pulmonary Research Laboratory, Charlotte Memorial Hospital and Mercy Hospital, Charlotte, N. C.

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Physical examination of the cardiovascular system showed moderate enlargement of the heart. The pulmonary second sound was somewhat accentuated and split. There was a systolic-diastolic thrill and a continuous systolic-diastolic machinery murmur in the left second and third interspaces parasternally. The pulse rate was 86 per minute; blood pressure in the left arm 158/65 mm. Hg. Peripheral pulses were easily palpable and there were no symptoms of congestive heart failure.

Routine laboratory findings were essentially negative. Chest x-ray examination showed increased pulmonary vascular markings. The heart was moderately enlarged, due primarily to left ventricular enlargement. There was also a dilatation of the ascending aorta and aortic arch. The pulmonary conus was prominent and pulsated expansively. Electrocardiogram showed depressed S-T segments in Leads I-III, negative T waves in Leads II-III, diphasic T waves in V_5-V_6. There were frequent ventricular extrasystoles.

Cardiac catheterization revealed evidence of a patent ductus arteriosus with significant left-to-right shunt (Fig. 1). The results were also suggestive of a moderate incompetence of the pulmonary valve.

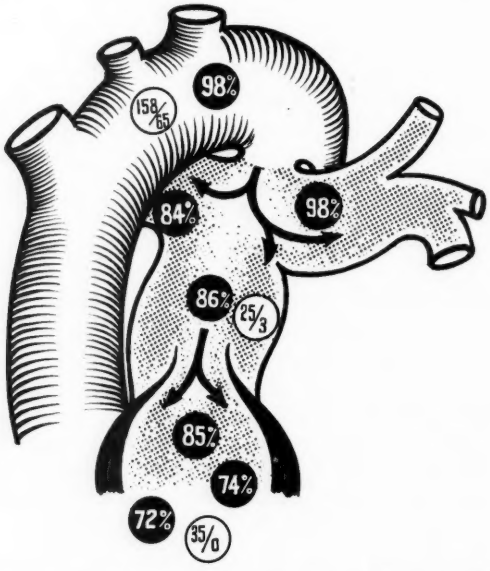

Fig. 1.—Schematic presentation of the preoperative hemodynamic conditions. The white circles show the blood pressure in mm. of mercury, the black circles the oxygen saturation of the blood.

At operation, a large patent ductus arteriosus (approximately 1 cm. in diameter) was found. This was occluded by two simple ligatures and multiple transfixion sutures. Immediately following the closure of the ductus, the anesthetist reported a 35 mm. Hg increase in the systolic and 40 mm. Hg increase in the diastolic blood pressure; otherwise the patient tolerated the procedure well. There were no changes in the force or rhythm of the heart action.

Two hours after operation the patient's blood pressure rose to 190/110 mm. Hg. The pulse rate increased to 140 per minute. Apresoline (25 mg.) and Thorazine (50 mg.) were administered, after which the blood pressure decreased to 160/90 mm. Hg, but within 4 hours it was elevated again to 210/120. The patient also had a protodiastolic gallop rhythm and complained of general discomfort. She had several attacks of paroxysmal

dyspnea. After 24 hours of continuous Apresoline and Thorazine therapy, she showed some improvement. The blood pressure remained relatively stabile between 170 to 160 systolic values, and dyspnea and orthopnea became less pronounced. The Apresoline and Thorazine were discontinued, but restarted after the blood pressure rose to 220/135 mm. Hg, and the patient developed a moderate attack of pulmonary edema. She remained on Apresoline, Thorazine, Hydryllin tablets, low sodium diet, and digitalis for 2 weeks. She was then discharged with a blood pressure of 175/100 mm. Hg and in relatively good condition.

Afterward the patient was closely followed. She reported several attacks of tachycardia, precordial pain, and paroxysmal dyspnea. The blood pressure varied between 145/90 and 190/110 mm. Hg. Peroral digitoxin annd Apresoline were continued. She is now able to perform limited physical activity.

CONCLUSIONS

The appearance of a severe systemic hypertension in the immediate postoperative period is an unusual complication following ligation of a patent ductus arteriosus. A reasonable explanation of this phenomenon is that outside of the possible increase in the blood volume[7-9] the patient had a narrowing of the systemic arterioles *before* surgery. However, the increase in the circulatory resistance did not manifest itself in hypertension because the systemic circulation was connected through the patent ductus with a low-pressure, large capacity vascular pool, i.e., the pulmonary circulatory system. Similar conditions have been created by the production of peripheral arteriovenous fistulae in clinical and experimental hypertension.[3, 6, 12, 13] When this patient's ductus arteriosus was occluded, the "latent" hypertension became manifest. This hypothesis is supported by the fact that the patient had a moderate elevation of the systemic blood pressure even in the presence of the patent ductus arteriosus. The preoperatively determined "corrected" systemic circulatory resistance was found to be elevated and gives additional support to the hypothesis.

The situation is very similar to that which exists in the presence of a patent duct with right-to-left shunt, the difference being that it is not the overloading of the pulmonary system but the systemic circulation which, in this instance, threatens the patient's life. From this case the conclusion may be drawn that in the presence of an elevation of the systemic arterial blood pressure, the ligation of a patent ductus should be performed with caution, as the occlusion of the "emergency vent" may lead to an increase of the blood pressure and a sudden overloading of the systemic circulation.

SUMMARY

The authors present a case of a 38-year-old woman with a patent ductus arteriosus accompanied by elevation of the systemic circulatory resistance and moderate increase in the systemic arterial blood pressure. The ligation of the ductus was complicated by an acute, severe systemic arterial hypertension.

An analogy is made between this condition and the situation that exists when a patent ductus arteriosus is occluded in the presence of pulmonary hypertension. The patent ductus arteriosus may serve in both instances as an emergency vent, and its occlusion may lead to serious consequences in either case.

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A METHOD FOR CONTROLLING A PATENT DUCTUS ARTERIOSUS DURING OPEN-HEART SURGERY

Philip Glotzer, M.D., and Allan E. Bloomberg, M.D.,
New York City, N. Y.

THE RECOGNITION and control of a patent ductus arteriosus during cardiac bypass with cardioplegia is essential for a successful run on the pump-oxygenator.[1] Failure to obtain control of the ductus results in massive and rapid distention of the right heart, severe pulmonary congestion, and death.

On occasion a patent ductus may be unrecognized because of misinterpretation of operative physical findings or be virtually impossible to control safely because of dense pericardial and pleural adhesions. We recently faced this situation, and herein is presented a method which allowed successful control of the ductus.

The patient was an 11½-year-old girl who was admitted to the hospital for the open-heart repair of a tetralogy of Fallot lesion. Seven years previously, this child had undergone a transventricular pulmonary valvulotomy at this hospital. At that time, a ductus had been looked for and not found. At this operation very dense pleural and pericardial adhesions were present. A thrill over the pulmonary artery was thought to be due to persistent pulmonic stenosis and pulmonic insufficiency.

The moment cardiac arrest was produced, a rapid dilatation of the right ventricle occurred. This was relieved by immediate right ventriculotomy. The resulting continuing blood loss was alarming because of the rapid fall in the reservoir level of the oxygenator. Control of the blood loss was then immediately obtained by occluding the pulmonic opening of the ductus with a finger passed into the pulmonary artery (Fig. 1). The blood level in the reservoir instantly stabilized. With the finger in place, the ventriculotomy was continued out into the pulmonary artery to the level of the ductus. A purse-string suture was placed around the ductus orifice, which resulted in complete control of the bleeding (Fig. 2). The operation then proceeded with a direct approach to the congenital intracardiac defects.

From the Surgical Division, The Montefiore Hospital, New York City.
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Fig. 1.—Control of the ductus is obtained by occluding the pulmonic orifice with the finger. The dotted line shows the proposed line of incision.

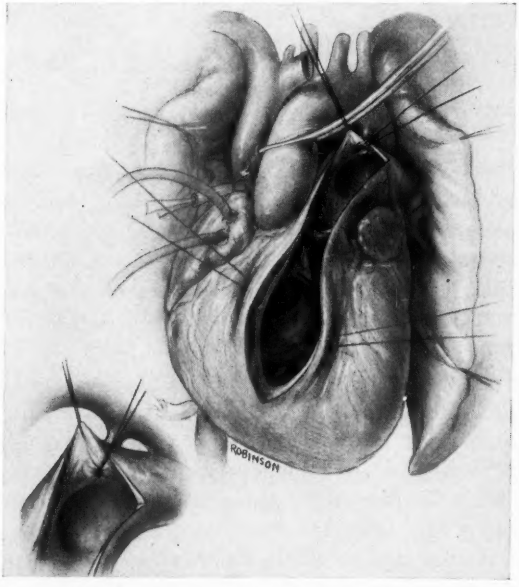

Fig. 2.—While maintaining control with the finger, a purse-string suture is placed around the pulmonic orifice. When the suture is tied, no further problem with the ductus exists.

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CONGENITAL DEFICIENCY OF THE PERICARDIUM

Alexander C. Hering, Commander (MC) USN, John S. Wilson, Lieutenant (MC) USN,** and Robert E. Ball, Jr., Lieutenant (MC) USN,****
Chelsea, Mass.

ALTHOUGH the 2 cases to be reported here apparently represent the one hundredth and one hundred first of a colorful series that began in 1559,[1] it is anticipated that with an increasing number of thoracotomies and constant improvement in radiologic techniques, the condition may soon forfeit its appellation of "a rarity."

Ellis, Leeds, and Himmelstein[1] have recently described 2 patients who had complete absence of the left parietal pericardium. The reader is referred with enthusiasm to their excellent article which includes interesting diagnostic techniques and a classification of pericardial defects.

As has been noted many times in the literature, pericardial defects are seldom diagnosed or encountered prior to autopsy. We would like to report 2 cases of partial absence of the left parietal pericardium in which the diagnosis was suggested before operation and confirmed by thoracotomy. It is our feeling that these cases demonstrate certain characteristic features which may be of value in the diagnosis and treatment of patients with pericardial defects.

CASE REPORTS

CASE 1.—A. H. W., 244120, a 22-year-old Negro man, was admitted to the U. S. Naval Hospital, Chelsea, Mass., on Feb. 14, 1958, for evaluation of a pulsatile mass along the upper third of the left cardiac border. Three years prior to admission a routine 70 mm. photofluorogram revealed an abnormal cardiac configuration in the then asymptomatic patient. Cardiac fluorography and radiography on June 27, 1955, revealed the heart to be globular in appearance with a definite increase in the size of the upper third of the left cardiac border which appeared to be pulsatile and was assumed to represent the pulmonary artery. No ventricular enlargement was noted, nor did the left atrium appear selectively enlarged. The aorta was reported as small and the pulmonary vasculature normal. No abnormal physical findings were recorded and the electrocardiogram was interpreted as being within normal limits. Following this examination the patient was returned to full duty aboard ship.

The occasion of his admission to this hospital was recurrent left anterior chest pain which was described as sharp and pressure-like, occurring both at rest and with exercise,

From the U. S. Naval Hospital, Chelsea, Mass.

The opinions herein expressed are those of the authors, and do not necessarily reflect the views of the Medical Department of the Navy or of the Naval Service at large.

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*Thoracic Surgeon.
**Senior Resident in Surgery.
***Senior Resident in Medicine.

A. *B.* *C.*

Fig. 1.—Case 1. *A*, Preoperative posteroanterior roentgenogram of the chest showing characteristic left atrial appendage shadow. *B*, Angiocardiographic exposure demonstrating the relationship of the pulmonary artery to the left atrial appendage. *C*, Postoperative posteroanterior roentgenogram of chest showing the persistence of the left atrial bulge minus the appendage.

nonradiating, and of about 2 minutes' duration. This complex occurred several times a month and had been noted for approximately 2 years. The pain was unrelated to food intake, activity, or position. Other and possibly related symptoms included mild dyspnea on climbing two flights of stairs and mild syncope during extreme heat, both symptoms relieved by rest. The balance of the history was noncontributory.

Physical examination revealed a well-developed, well-nourished Negro man in no distress. Blood pressure was 126/72 mm. Hg, pulse 84, respirations 14, and temperature 98.6° F. Peripheral pulses were readily palpable and equal bilaterally, and there was no cyanosis, clubbing, or edema. A mild, left sternal border heave was palpable along with a tapping apical impulse, the point of maximum intensity being indistinct. Cardiomegaly was not present. The rate was 84 with a variable sinus arrhythmia and occasional premature contractions. A Grade 2 (of 6) blowing systolic murmur was audible along the left sternal border, maximal at the third intercostal space. A precordial friction-type sound could be heard on occasion with the patient standing and flexed 45 degrees at the waist. The remainder of the physical examination was entirely normal.

Laboratory studies, including blood chemistry, hemogram, urinalysis, serologic test for syphilis, C-reactive protein, anti-streptolysin-O titer, skin tests for tuberculosis and mycotic disease, and sickle cell preparations, were within normal limits. Electrocardiograms revealed frequent ventricular ectopic beats, some of which were not premature and suggestive of a sinus pause and ventricular escape beat, but were regarded as within normal limits.

Cardiac fluoroscopy again revealed a prominent bulge along the left upper heart shadow which had not apparently changed since the initial examination recorded in 1955. Expansile pulsation of the abnormal bulge was noted, but no cardiac chamber enlargement could be demonstrated in the examination made after a barium swallow.

On March 3, 1958, right heart catheterization was performed, and this revealed normal right cardiac hemodynamics without evidence of shunt. All accompanying determinations done during this study were within normal limits. On March 26, 1958, angiocardiograms were made in the posteroanterior and oblique projections with the use of 70 per cent Diodrast injected in the basilic vein through a Robb-Steinberg needle. The pulmonary artery and aorta could be definitely excluded as contributing to the abnormal shadow, but suspicion was cast upon the left pulmonary veins or an abnormally placed left atrial appendage.

The patient was then observed in several of his "attacks" during which he became faint, perspiring, and complained bitterly of pain in the left anterior chest. At the Cardiovascular-thoracic Conference, thoracotomy was advised.

On June 13, 1958, a left thoracotomy was performed through the left fifth interspace. The left upper lobe was found to be adherent to the left atrial appendage by firm adhesions, apparently of long standing, with some recent inflammation. The left atrium and its appendage lay well outside the normal confines of the cardiac mass. The appendage was elongated and sigmoid in shape. The pericardium was absent from the atrioventricular groove cephalad, and the cephalic margin was rolled and thickened. With each cardiac contraction the rolled margin massaged the left coronary and circumflex vessels. The pericardium, which was present, resembled an egg-cup, and the heart, the egg (see Fig. 3). There was no free left pericardial cavity, the remaining pericardium being adherent by fine but firm adhesions to the epicardium. Adhesions were divided and ligated where necessary. The left heart was explored digitally through an incision in the left atrial appendage. No abnormalities were detected by this maneuver. The appendage was amputated and the margins closed with interrupted and continuous silk sutures. Because of the anatomic position of the rolled pericardial edge and the adherent pericardium, a generous V-shaped excision of the pericardium over the right and left ventricles was performed. The patient recovered from the operative procedure without complication or difficulty. He stated during his postoperative course that the chest pains were no longer present. One year following operation he is well and working.

CASE 2.—S. A. C., 253921, a 21-year-old sergeant in the USMC was admitted to the U. S. Naval Hospital, Chelsea, Mass., on June 1, 1959, with an undetermined diagnosis of

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A. *B.* *C.*

Fig. 2.—Case 2. *A*, Preoperative posteroanterior roentgenogram of the chest showing characteristic left atrial appendage shadow. *B*, Angiocardiographic exposure showing the relationship of the pulmonary artery to the left atrial appendage. *C*, Angiocardiographic exposure made during the left heart filling phase showing outlining of the left atrial appendage.

dilatation of the pulmonary artery. This diagnosis was entertained following a routine chest x-ray examination which was reported as showing an abnormal pulmonary conus. He was entirely without symptoms, and physical examination was within normal limits. He was referred to the hospital for further studies referable to the abnormal shadow. This shadow consisted of a rather angular projection just lateral and inferior to the pulmonary artery, in the position of the left atrial appendage. In view of the previous case, a tentative diagnosis was made of herniated left atrial appendage due to partial absence of the pericardium.

Routine laboratory studies and further films of the chest exposed during barium swallow were not unusual. Angiocardiography with 70 per cent Sodium Urokon demonstrated that the pulmonary artery was innocent, but that the left atrial appendage became opacified at the proper interval and occupied the same position as the abnormal shadow noted on the plain films. At the Cardiovascular-thoracic Conference it was the consensus that although deficient pericardium was the most likely possibility, thoracotomy should be resorted to in order to rule out other lesions in this area.

On June 25, 1959, an exploratory thoracotomy was performed through the left fifth interspace, sparing the trapezius and rhomboid muscles. The ribs were spread just sufficiently to permit adequate visualization and palpation of the hilum and adjacent cardiac structures. As anticipated (see Fig. 4), the left atrial appendage resided partly outside the main cardiac mass, herniating through an upper pericardial window, and performing a considerable excursion with each cardiac systole. The left phrenic nerve comprised the medial margin of the pericardial defect. There were no adhesions, and the finger passed readily into the free pericardial cavity. Since the aperture seemed large enough to permit free action of the left atrial appendage and atrium, and too small to permit herniation or strangulation of the atrium or ventricle, no attempts were made to correct the condition, and the chest was closed. Postoperatively there were no complications, and the patient was discharged to duty July 28, 1959. Four months postoperatively he was having no difficulty.

EMBRYOLOGY

Studies by previous authors of the embryological antecedents of pericardial malformations have for the most part tended to incriminate the formation of the pleuropericardial and pleuroperitoneal folds. We agree with this concept and cite the case of Ladd[2] in its support. Ladd found a hernia of the Bochdalek type occurring in a patient who had deficiency of the pericardium. These lesions certainly involve the pleuroperitoneal and pleuropericardial folds. The theory has been entertained in addition that the growing lung pushes its way into the area deficient of pericardium, and so prevents the complete development of the pericardium. Our first case with lung firmly adherent to the heart may or may not bear out this latter concept.

CLINICAL AND DIAGNOSTIC FEATURES

It will be noted that Case 1 (Fig. 3), who had the larger defect and extensive inflammatory adhesions, had recurrent chest pain, and, at times, a pleuropericardial sound could be heard. The second patient, who had a smaller defect and no adhesions, was asymptomatic (Fig. 4). Right heart catheterization, having been normal in the first case, was not thought to be necessary in the second. Although we did not make use of diagnostic pneumothorax in either patient, we believe that it may be a valuable aid. In one postoperative film of the chest in the second patient, a small amount of right pericardial air may be seen, suggesting that use of preoperative pneumothorax would have been helpful in diagnosis.[1]

In Case 2 (Fig. 2, *B* and *C*), angiocardiography was diagnostic. Those in Case 1 were less clear, but on careful inspection we felt that they were quite suggestive of the diagnosis in spite of technical factors producing a poor left-side filling phase.

Fig. 3

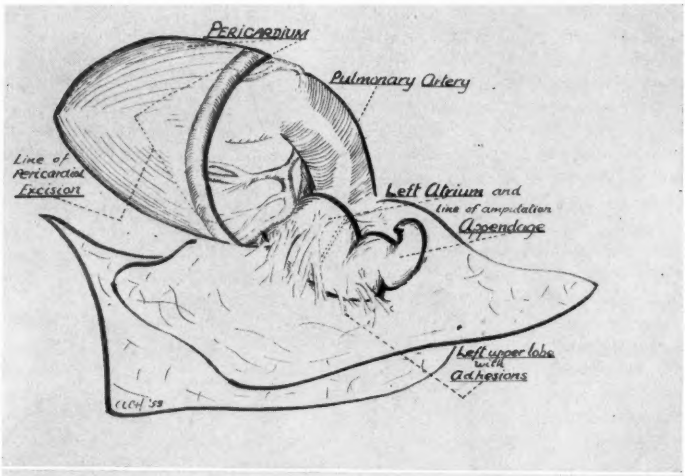

Fig. 4.

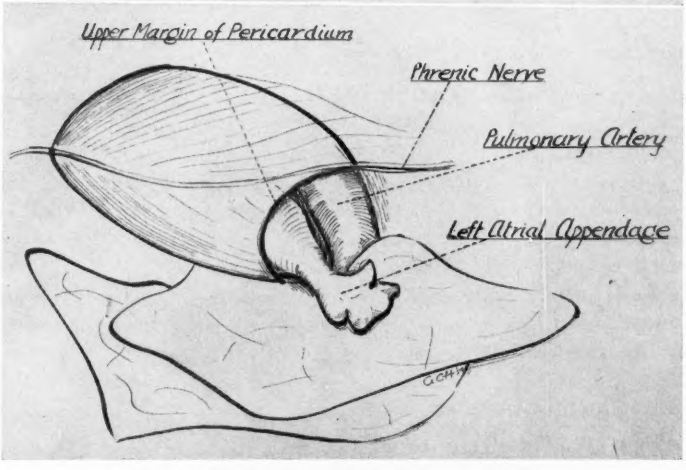

Fig. 3.—Sketch of operative findings (Case 1).
Fig. 4.—Sketch of operative findings (Case 2).

SURGICAL ASPECTS

In Figs. 3 and 4 may be found the operative findings as drawn by the operator. Some consideration was given to the question of amputating the herniating left atrial appendage in the second case. Marked cardiac irregularity occurred when a clamp was applied to the base of the appendage, and the idea was forthwith abandoned.

CONCLUSIONS

1. It is our opinion, after a survey of the literature and the study of 2 patients who had similar diagnostic and surgical features, that congenital deficiency of the pericardium may be diagnosed with the aid of angiocardiography and pneumothorax.

2. Characteristic features in partial absence of the left pericardium are primarily radiologic, namely, an abnormal bulge in the region of the left atrial appendage in the standard posteroanterior roentgenogram, and the outlining of the appendage with contrast medium during the left heart filling phase of an angiocardiogram.

3. If the diagnosis seems clear, and the patient is, and has been, asymptomatic, surgical intervention may not be necessary. If symptoms are present, relief may follow thoracotomy, where atrial appendectomy, division of adhesions, and pericardioplasty may be performed.

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REPLACEMENT OF THE MAIN PULMONARY ARTERY BIFURCATION BY AUTOGENOUS PERICARDIUM

Lester R. Sauvage, M.D., Abraham M. Rudolph, M.D.,** and Robert E. Gross, M.D., Boston, Mass.*

A TECHNIQUE has been developed for the successful one-stage experimental replacement of the distal main pulmonary artery, its bifurcation, and the major portions of both right and left pulmonary arteries that does not require the use of pumps, extracorporeal circulation, hypothermia, or circulatory interruption. This method was developed to allow for the study of pericardial autografts in the pulmonary arterial system. In this paper we shall outline the details of graft placement and shall describe the status of two such grafts over observation periods of 16 and 17 months, respectively.

METHOD

The method for extensive pulmonary artery resection and pericardial autograft replacement is shown in Fig. 1. The key step in this experimental technique is the transplantation of the left pulmonary artery to a position adjacent to the valve without producing stenosis of its orifice. We attempted this procedure in 6 adult mongrel dogs (average weight, 20 kilograms) with 2 survivors. The cause of death in the 4 dogs that died during the operation was acute right heart failure secondary to stenosis of the orifice of the transposed left pulmonary artery. Both of the dogs that survived operation were studied postoperatively by right heart catheterization and angiocardiography, one at 6 and 13 months and the other at 9 months after operation.

RESULTS

Right heart catheterization in both dogs at the postoperative intervals mentioned above revealed a peak systolic pressure in the right ventricle of about 40 mm. Hg. The angiocardiographic studies revealed no evidence of aneurysm

From the Department of Surgery, The Children's Medical Center, and the Harvard Medical School.

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*Trainee, National Heart Institute.

**Established Investigator, American Heart Association.

formation or of significant contracture in either graft (Figs. 2 and 3). One of the 2 dogs that survived operation died at 17 months after operation, while the other dog is in good condition at 16 months after operation. The dog that died developed moderate ascites at 12 months but remained active up to the day of

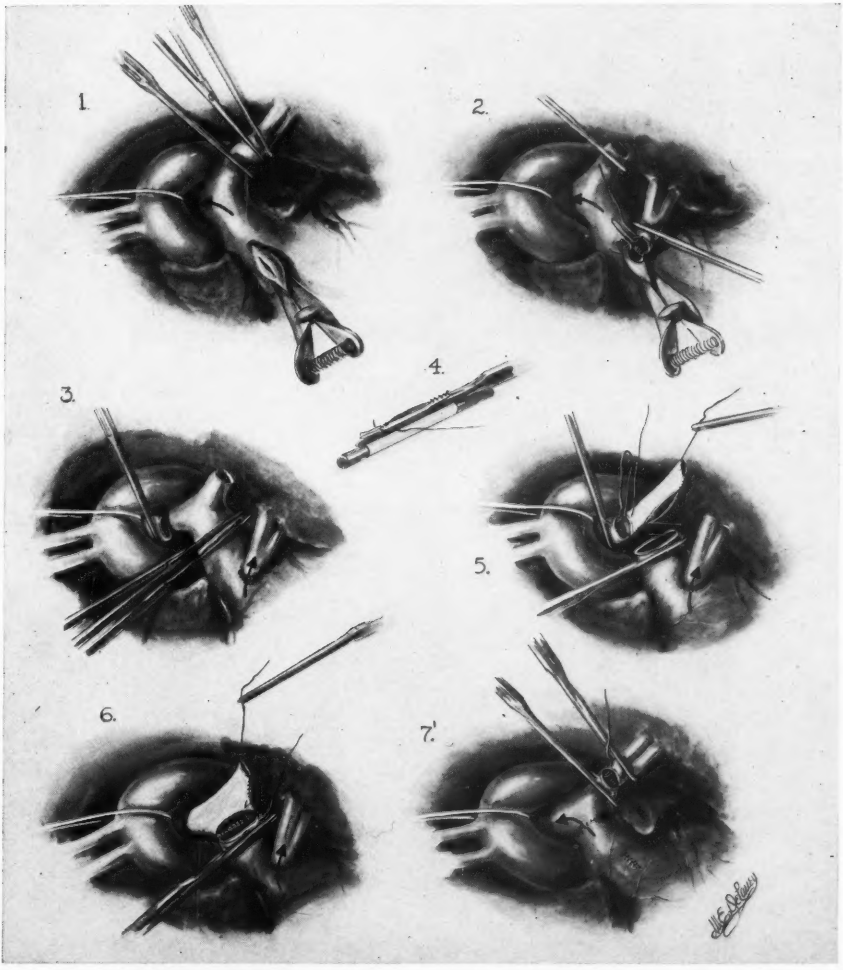

Fig. 1.—Technique for the experimental resection and replacement of the distal main pulmonary artery, its bifurcation, and the major portions of both right and left pulmonary arteries by a pericardial autograft.

death. The repeat studies of this dog at 13 months (Fig. 3) did not substantiate our clinical impression that the ascites was secondary to contracture of the graft. Autopsy examination revealed moderate ascites, hepatic congestion, an enlarged right atrium and right ventricle, tricuspid insufficiency, and slight narrowing

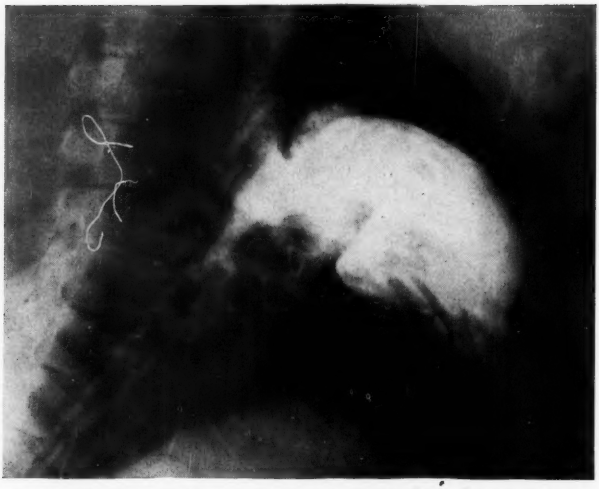

Fig. 2.—Angiocardiogram made 9 months postoperatively, in right lateral projection, of dog with pericardial autograft replacement of the distal main pulmonary artery, its bifurcation, and the major portions of both right and left pulmonary arteries. Ninety per cent Hypaque was injected through a catheter positioned in the right external jugular vein. Note the opacified superior vena cava, right atrium, right ventricle, and the pulmonary arterial tree. There is neither aneurysmal dilatation nor significant contracture of the graft. The shadow visible above the right ventricular outflow tract is the normal-sized appendage of the right atrium. Right heart catheterization revealed a peak systolic pressure of 40 mm. Hg in the right ventricle and a gradient across the graft of 20 mm. Hg.

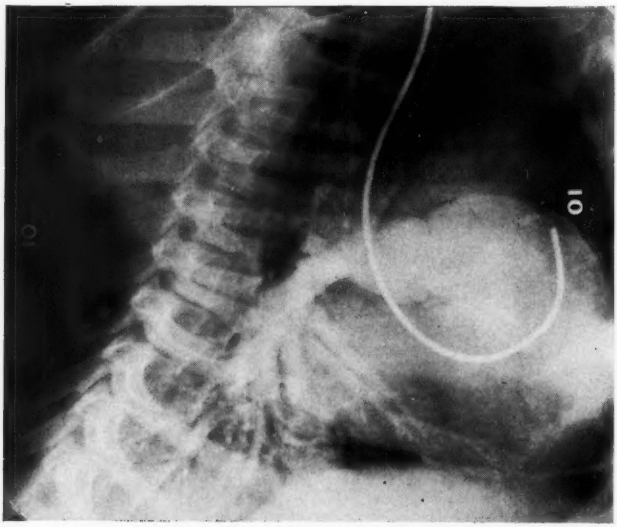

Fig. 3.—Angiocardiogram made 13 months postoperatively, in right lateral projection, of dog with pericardial autograft replacement of the distal main pulmonary artery, its bifurcation, and the major portions of both right and left pulmonary arteries. Ninety per cent Hypaque was injected through the catheter which had been passed from the right external jugular vein down the superior vena cava into the right ventricle. Note the absence of aneurysmal dilatation or of significant contracture in the graft. The shadow visible above the right ventricular outflow tract is the enlarged appendage of the right atrium. Right heart catheterization revealed a peak systolic pressure of 40 mm. Hg in the right ventricle and a gradient across the graft of 20 mm. Hg. Seven months previously the pressure findings at right heart catheterization were identical. However, the angiocardiograms at that time revealed a lesser degree of right atrial enlargement.

Fig. 4.—Roentgenogram, posteroanterior view, of the pulmonary arterial system of the dog dead at 17 months (512 days) after operation. The vessels are inflated to approximately 30 mm. Hg with air via the cannula positioned in the proximal main pulmonary artery. The outflow tract of the right ventricle has been separated from the rest of the heart. The trachea and major bronchi have been excised. The pulmonary veins are ligated at the lobar level. Note the absence of any evidence of calcification. The dimensions of the graft as measured on this roentgenogram are about 25 per cent less than those taken at the time of operation.

Fig. 5.—Photograph of specimen, the roentgenogram of which is seen in Fig. 4. This specimen has been opened from the posterior aspect. Note the appearance of the graft. Its inner surface is smooth and glistening. The suture lines are well healed and the walls of the graft are soft and pliable. The vertical suture line in the main pulmonary artery adjacent to the valve is the site where the left pulmonary artery had been anastomosed during implantation of the graft (see Fig. 1).

of the graft (Fig. 4). The graft was soft, pliable, and free of degenerative change. Its inner surface was smooth and glistening. The multiple suture lines were well healed (Fig. 5). There was no evidence of pulmonary infarction.

DISCUSSION

The method described in this paper for resection and replacement of the main pulmonary artery bifurcation is technically difficult of accomplishment. However, the technique could be simplified by interposing a temporary graft between the end of the left pulmonary artery and the right ventricle.

We are greatly interested in the fate of autogenous pericardium in the pulmonary arterial circuit. Robinson, Glotzer, Gilbert, and Hurwitt[1] have shown that aortic homografts in the pulmonary artery are subject to severe degenerative change in the pericardial autograft, reported here to be significant at 17 months (Figs. 4 and 5). In other experimental studies in the dog we have observed pericardial autografts to function well and to show no evidence of degenerative change in the intrathoracic inferior vena cava (Sauvage and Gross[2]) and in the right ventricular outflow tract (Sauvage, Gross, Rudolph, Pontius, and Watkins[3]).

On the basis of these observations and those reported here we are employing autogenous pericardium clinically for widening of the right ventricular outflow tract and of the pulmonary artery in instances of infundibular stenosis or hypoplasia of the artery. Longer periods of observation are required to ascertain the ultimate fate of autogenous pericardium in the pulmonary artery. We are keeping the remaining animal reported in this paper for such purpose.

SUMMARY

A technique for the experimental resection and replacement of the distal main pulmonary artery, its bifurcation, and the major portions of both right and left pulmonary arteries by autogenous pericardium has been presented. We believe this technique of graft implantation to be a useful experimental method, and the satisfactory function of two such pericardial autografts over implantation periods of 16 and 17 months, respectively, to be of clinical significance.

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EXPERIMENTAL REPLACEMENT OF THE AORTIC ARCH BY HOMOGRAFTS

Lester R. Sauvage, M.D., Abraham M. Rudolph, M.D., and Robert E. Gross, M.D., Boston, Mass.*

THIS PAPER has the following three purposes: first, to outline a simplified method for the experimental replacement of the aortic arch and its branches; second, to describe the function over the first year of two homografts so implanted as aortic arch replacements; and third, to present observations of the responsiveness of the cardiovascular system to various stimuli after replacement of the aortic arch.

EXPERIMENTAL METHOD

Six adult mongrel dogs were employed in this study. Each dog was given 30 mg. of sodium pentobarbital per kilogram of body weight intravenously. Hypothermia was induced by partial immersion of the anesthetized animal in cold running tap water for from 10 to 20 minutes (Sauvage, Pontius, Watkins, and Gross[3]). After opening the left hemithorax through the fourth intercostal space, the aortic arch was resected and continuity re-established with an arch homograft by the method depicted in Fig. 1. The key step in this method is that shown in step *3* in which the azygos vein is allowed to drain into the heart while the venae cavae are occluded. Thus, while aortic continuity is interrupted, the coronary circulation is maintained by the azygos inflow into the heart. After occluding the venae cavae, the heart should be allowed to contract several times before the ascending aorta is clamped in order to avoid the development of excessive intracardiac pressures.

Postoperatively, the 2 surviving animals were observed carefully and each was studied by aortography, 1 at 164 days and the other at 178 days after operation. The response of the blood pressure and heart rate to epinephrine, norepinephrine, serotonin, atropine, anoxia, and hypercarbia were determined in 1 of these 2 dogs at 214 days after operation.

RESULTS

Four of the 6 dogs undergoing the procedure illustrated in Fig. 1 died because of problems related either to the technique of implantation or to the

From the Department of Surgery, The Children's Medical Center, and the Harvard Medical School.

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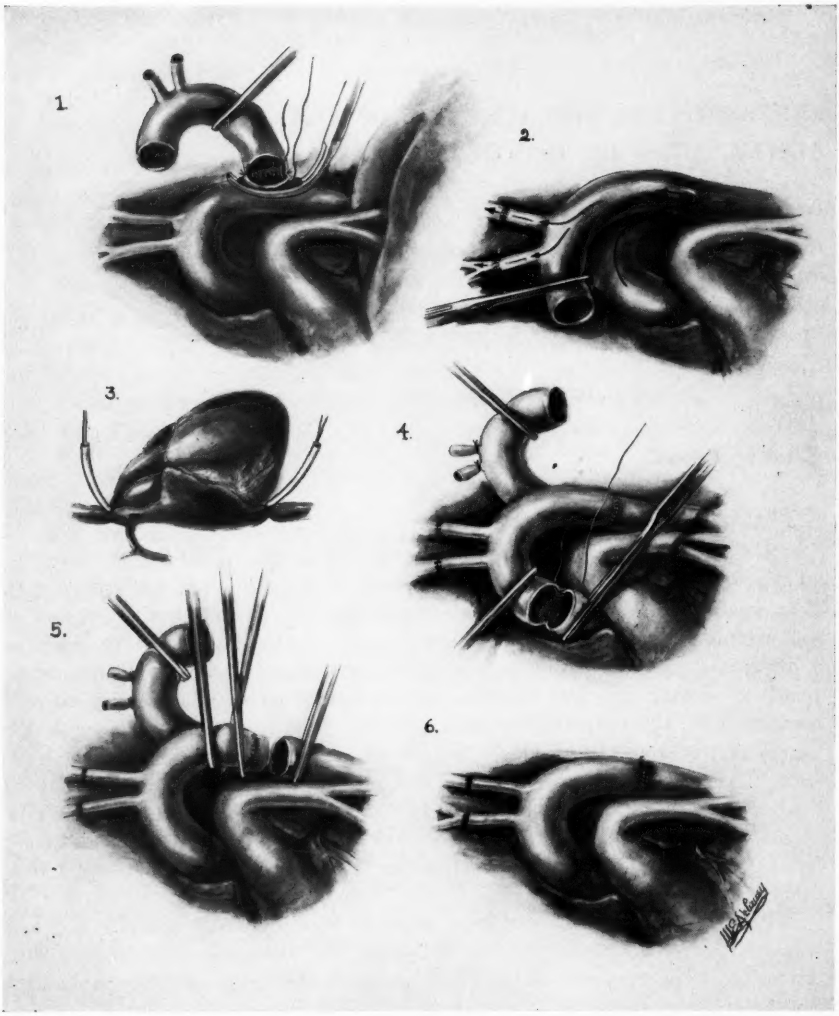

Fig. 1.—Method of resecting the aortic arch and restoring continuity by means of an aortic arch homograft. The graft is first anastomosed to the side of the descending thoracic aorta as shown in step *1*. Then the left brachial artery (subclavian) of the dog is divided and its distal end anastomosed to the brachial artery branch of the graft. The graft is then occluded between the origins of its left brachial and brachiocephalic branches. The clamps are then removed from the brachial artery and the aorta of the dog. This re-established circulation through the left brachial artery by means of a retrograde endograft shunt. A similar procedure is then performed for the anastomosis of the brachiocephalic artery to the graft. The completed anastomoses for these two branches are shown in step *2*. In step *3* the venae cavae are occluded while the azygos vein is left open. The heart is allowed to contract several times to deplete the cardiopulmonary circuit of blood before the ascending aorta is clamped. The anastomoses of the graft to the ascending aorta is shown in step *4*. The coronary circulation is maintained throughout this step by virtue of the azygos vein which was left open in step *3*. Following completion of step *4*, the end-to-side anastomosis of the graft to the descending aorta is taken down (step *5*) and an end-to-end union effected (step *6*).

development of deep hypothermia. Both of these complications are easily avoidable as experience is acquired with the method. Neither of the 2 dogs that survived this procedure received intravenous fluids or blood transfusions. Both of these animals were rendered moderately hypothermic by immersion in cold running tap water according to the method referred to above.

The first surviving dog, a 16 kilogram animal, after 15 minutes in running tap water at 12° C. had a rectal temperature of 30° C. At the end of operation, the rectal temperature was 28° C. The ascending aorta was occluded for 12 minutes while the graft was anastomosed to the proximal end of the ascending aorta (step *4*, Fig. 1). The heart did not miss a beat during this period of occlusion of the ascending aorta; its contractions remained vigorous throughout and its color pink. This animal recovered normally and manifested no evidence of neurologic damage. The aortogram of this dog at 178 days after operation is shown in Fig. 2. The blood pressure and heart rate responses of this animal to epinephrine, norepinephrine, serotonin, atropine, anoxia, and hypercarbia were studied at 214 days postoperatively and found to be normal. This animal continues to be in excellent general condition at 14 months after operation.

The second surviving animal, an 11 kilogram dog, after 10 minutes in running tap water at 10° C. had a rectal temperature of 32° C. At the end of operation, the rectal temperature was 25° C. The ascending aorta was occluded for 7 minutes while the graft was anastomosed to the proximal end of the ascending aorta (step *4*, Fig. 1). The heart beat strongly throughout the period of occlusion of the ascending aorta, but fibrillation ensued shortly after removal of the occluding clamp as the heart was being displaced from its normal position to allow for removal of blood from the pericardial cavity. However, defibrillation was easily accomplished by one countershock. This animal recovered normally and manifested no evidence of neurologic damage. The aortogram at 164 days after operation is shown in Fig. 3. This animal is now 13 months postoperative and continues to be in excellent general condition.

DISCUSSION

Sauvage and Wesolowski[4] observed that Orlon prostheses were unsuited for use as aortic arch replacements because of kinking in regions of angulation. These grafts were implanted in normothermic animals using a technique that employed retrograde endograft shunts (steps *1* and *2*, Fig. 1) and a shunt from the left ventricle into a side arm of the graft while the prosthesis was anastomosed to the proximal end of the ascending aorta. Satinsky, Neptune, and Alai[2] reported an impressive series of successful homologous transplants of the aortic arch in dogs employing a similar technique. However, the method reported here has seemed easier to perform and, therefore, would appear to have merit as a procedure for laboratory use.

The many complexities of the architecture of the aortic arch have stymied the manufacture of a prosthesis suitable for replacement of this portion of the aorta. It seems probable that aortic arch homografts will continue to be the

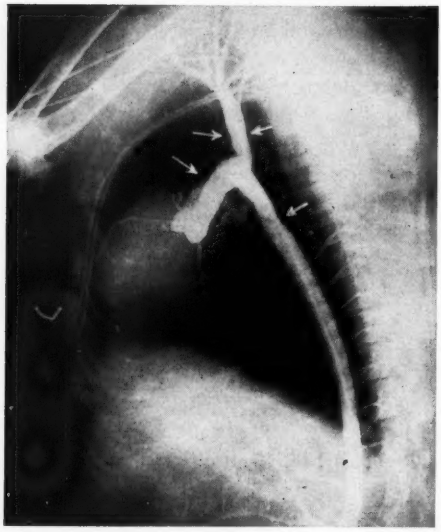

Fig. 2.—Aortogram made 178 days after operation which shows the aortic arch homograft to be functioning satisfactorily. The anastomoses are indicated by arrows. (This graft had been preserved 5 days in saline solution at 4° C. prior to implantation.)

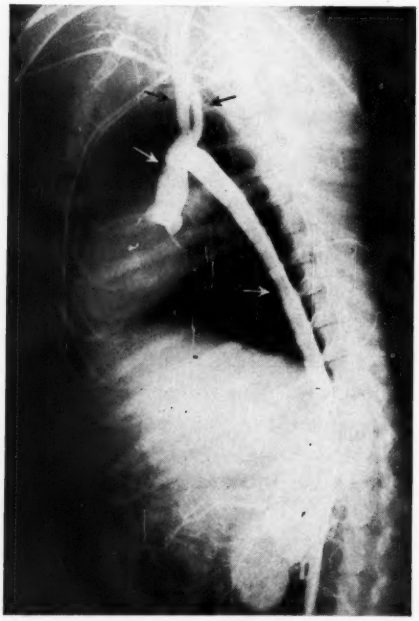

Fig. 3.—Aortogram made 164 days after operation which shows the aortic arch homograft to be functioning satisfactorily. The anastomoses are indicated by arrows. (The graft was 1 hour old at time of implantation.)

preferable type of graft in this location for many years. De Bakey, Crawford, Cooley, and Morris[1] reported the use of a homograft for the first successful clinical replacement of the aortic arch. Extracorporeal circulation was employed to maintain essential blood flow during the period of interruption of aortic continuity. However, little is known of the long-term fate of homografts employed for replacement of the aortic arch. The 2 dogs reported in this paper are being kept for such study and will be reported subsequently.

It is of interest that the response of the cardiovascular system to epinephrine, norepinephrine, serotonin, atropine, anoxia, and hypercarbia was normal in the 1 dog so studied at 214 days after aortic arch resection and homograft replacement. The aortogram of this animal is shown in Fig. 2. It is probable that carotid sinus mechanisms were able to compensate for the loss of the pressor and chemoreceptor mechanisms contained in the aortic arch. Whether similar compensation occurs in the human being is not known.

SUMMARY

An experimental method has been described for replacing the aortic arch that employs retrograde endograft shunts and moderate hypothermia in which cardiac ischemia is avoided by leaving the azygos vein open during the period of vena caval inflow stasis. This technique is recommended for the laboratory study of aortic arch grafts because of its comparative simplicity.

Aortograms at 6 months after operation and clinical observation for 1 year of the 2 dogs in this study indicate excellent function during this implantation period of the homografts employed as aortic arch replacements.

Studies were conducted in 1 of these 2 animals of the responsiveness of the cardiovascular system to a variety of stimuli. The results indicate that replacement of the aortic arch in the dog is not associated with any significant change in cardiovascular reactivity.

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SOME HELPFUL INSTRUMENTS FOR CARDIOVASCULAR SURGERY

Albert Welberry, Ph.D., Elliott Senderoff, M.D., Isadore Kreel, M.D., and Ivan D. Baronofsky, M.D., New York, N. Y.*

I. ATRIAL CLAMP WHICH ALLOWS FOR REPEATED RE-ENTERING OF ATRIUM

ADVANCES in open-heart surgery have necessitated the creation of new instruments designed to meet the special requirements of this field. The clamp shown in Fig. 1 is designed to allow for extensive surgery within the atrial cavity by providing maximum exposure with complete control of the wound. The clamp allows the surgeon to stop at any point in the operative procedure and easily and hemostatically approximate the wound edges by simply approximating the jaws of the clamp. This enables the operator to open and close the atrium and to enter and re-enter the heart as frequently as he desires. In cases performed under complete occlusion of the cardiac inflow, the clamp is of value in extending the operating time by allowing the surgeon to close the atrium, allow a temporary period of cardiac inflow, and then re-enter the atrial cavity to continue the procedure under inflow occlusion. The surgeon is able also to suture the atriotomy wound leisurely in a dry field with no tension on the sutures, as the wound edges are approximated by the clamp when this procedure is performed.

The instrument is a conventional cardiovascular clamp with atraumatic blades. On the outer surface of each blade are three small cross-bars. On the handle is a ratchet which prevents accidental opening of the clamp. There is also a second ratchet which allows the blades to be fixed in the open position. The latter ratchet is controlled by a side bar which can be easily manipulated by the finger. To use this instrument, two parallel rows of guide sutures are placed along the anterolateral wall of the atrium along the line of the projected atriotomy. Each row should contain three sutures. All the sutures are then elevated and the sutures and intervening atrial tissue clamped. The sutures are then tied snugly to the small cross-bars on the outside of the clamp, and the atriotomy made between the two rows of sutures (Fig. 2, *A*).

From the Division of Experimental Surgery, The Department of Surgery, The Mount Sinai Hospital, New York, N. Y.

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*Fellow, New York Heart Association.

The clamp is then opened widely and locked in the open position with the side bar (Fig. 2, *B*). This presents a large rectangular opening in the atrium with excellent exposure of all parts of the atrial chamber. If further exposure is needed, the atriotomy is extended. At the completion of the procedure, air is flushed out of the atrial cavity and the atriotomy rapidly sealed by approximating the clamp blades. If the atriotomy has been extended, the end of the incision is brought between the blades by elevating it with a forceps. The atriotomy may then be subsequently sutured at leisure (Fig. 2, *C*).

The clamp described has had extensive trial in the experimental direct vision production of interatrial septal defects and has been very satisfactory.

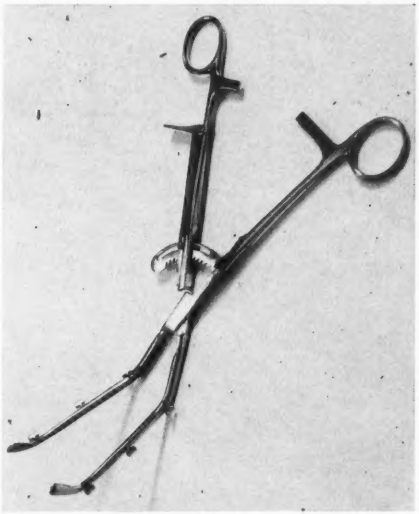

Fig. 1.—Atriotomy clamp.

It is projected for clinical use in the open repair of such defects. The clamp has also been successfully used in the surgical creation of complete heart block in 5 dogs[1] utilizing complete occlusion of the cardiac inflow without hypothermia or cardiac bypass. The clamp permitted repeated entry into the atrial cavity until a ligature effectively producing complete heart block was placed.

The clamp can be made in two angles. The one shown (Fig. 1) is more effective in dog experimentation because of the shallow thoracic cavity. A more acute angle is necessary for the deeper chest of the human being.

II. SUCTION TIP FOR LARGE AND SMALL BLOOD LOSS

The need for adequate suction to maintain a clean operative field is of extreme importance in intracardiac surgery. This is often difficult to achieve despite the varied types of suction units presently available. The unit shown in Fig. 3, *A* has been designed to provide effective and efficient suction of both

large and small amounts of fluid. The unit is available in three sizes with internal diameters ranging from 3 to 6 mm. Despite this large inflow capacity, the design is such that the suction tip is not bulky and cumbersome. The instrument shown disassembled (Fig. 3, *B*) consists of an inner tubular shaft, an outer shaft, a tip consisting of four lateral holes and one distal hole, a spring controlled lever, and a handle. In the resting position (Fig. 4, *A* and *B*),

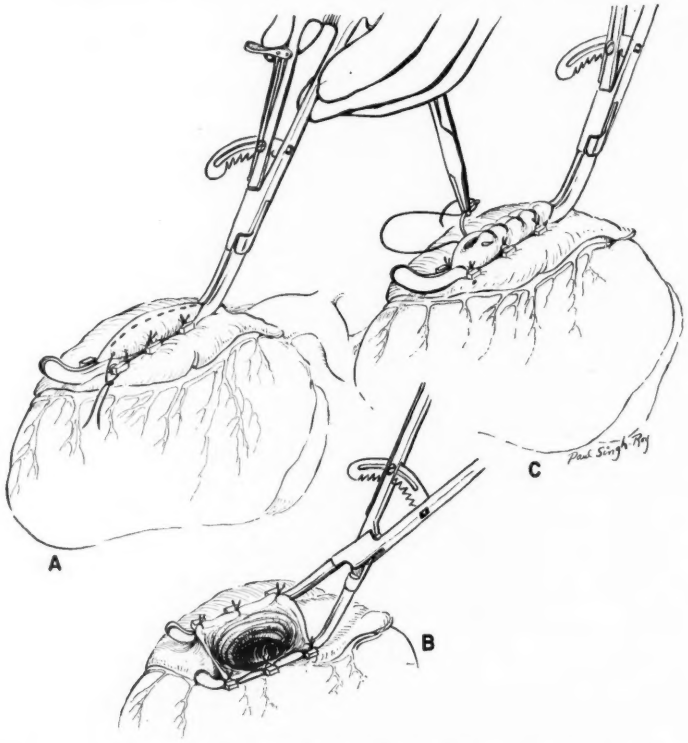

Fig. 2.—*A*, Atriotomy clamp in position on wall of atrium. Note sutures tied about the cross-bars of the clamp securing the atrial wall to the clamp blades. Dotted line indicates line of proposed incision in the tissue between the clamp blades.

B, Clamp locked in the open position. The wound edges and the extent of the wound opening are completely controlled by the clamp blades.

C, Clamp closed at the end of the operative procedure. The atriotomy incision can be sutured at leisure in a dry field.

all the inlet orifices are open and large amounts of fluid (1,150 to 2,400 c.c. per minute, depending upon size of suction unit) can be easily handled. This unit is also capable of handling the remaining small pools and drops of blood which tend to obscure the operative field. In most suction units, when an attempt is made to remove a small pool or drop of fluid, air enters the inlets before the fluid does. This breaks the effective vacuum and the suction tip does not function. In the unit described, upon depressing the lever, the side inlets are shut off (Fig. 4, *C*) and only the distal orifice of the suction tip is operative. This allows the

suction to be concentrated at the end of the tip only. In this manner the smallest drop of fluid can be removed from the field. When flooding occurs, the lever is released, the spring forces the handle up and the suction unit is once again able to handle large quantities of fluid. When dealing with small amounts

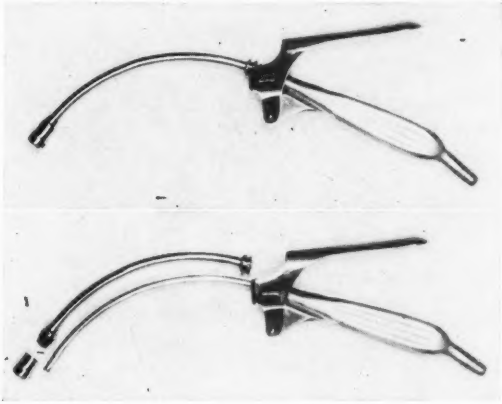

Fig. 3.—Suction unit. *B*, Suction unit disassembled.

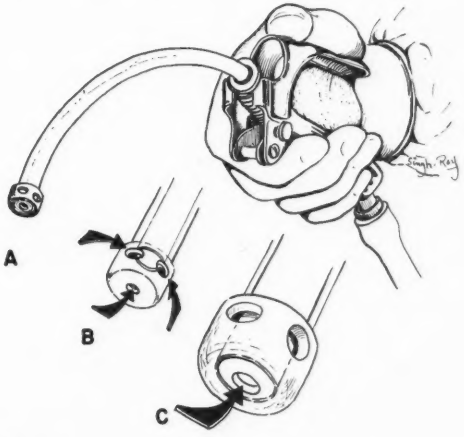

Fig. 4.—*A*, Suction unit.

B, Drawing of tip of suction unit showing side and distal inlet orifices operative in the usual resting position of the inner shaft.

C, Drawing of tip of suction unit when lever on handle is depressed. Inner shaft has been advanced to meet the distal orifice. This closes the side inlets and causes the suction to be operative at the distal orifice only.

of blood, this unit also decreases turbulence of the stream and the subsequent hemolysis of red blood cells as air is not able to mix with the blood when the lever is depressed. Since the suctioned blood is recirculated in cardiac bypass operations, it is important and advantageous to have a minimum of hemolysis occur as a result of the suction unit employed.

SUMMARY

Two new instruments* for use in the field of intracardiac surgery have been presented. A clamp for easy and effective control of atriotomy incisions and a suction unit capable of handling fluid volumes ranging from minute to large amounts with a minimum of hemolysis have been described.

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*The instruments described are manufactured by Edward Weck & Co., Inc., 135 Johnson Street, Brooklyn 1, N. Y.

BRONCHIECTASIS: RESULTS OF PULMONARY RESECTION

Lieutenant Colonel Thomas H. Hewlett, MC, USA, Fort Sam Houston, Texas, and Lieutenant Colonel H. Haskell Ziperman, MC, USA,** San Francisco, Calif.*

THE FREQUENCY of bronchiectasis has been emphasized in reports originating from hospitals which are devoted primarily to the management of pulmonary disease. The true incidence of this disease in the general population has not been clarified. Proved bronchiectasis is a surgical problem from the standpoint of either palliation or cure, and a significant period of time is devoted to the diagnostic investigation of suspect patients. Evaluation of the problem as it exists in the population served by a general hospital could be expected to clarify the incidence of the disease, as well as to confirm the results of surgical treatment.

The period 1949 to 1958 was chosen for study. All proved cases of bronchiectasis studied or treated by the Thoracic Surgical Service of Brooke Army Hospital are herein reported.

The yearly summary reports were utilized in deriving average figures in several categories. Excluded from the admission figures are (1) births, and (2) patients with proved diagnosis of active pulmonary tuberculosis.

TABLE I. ADMISSIONS

HOSPITAL ADMISSIONS	PATIENTS WITH PULMONARY DISEASE	CLINICALLY DIAGNOSED AS BRONCHIECTASIS	BRONCHOGRAPHY PERFORMED	PROVED BRONCHIECTASIS
20,792	560	61	85	11.5
Incidence of Bronchiectasis				
per 1,000 admissions			0.55	
per 100 patients with pulmonary disease			2.05	
per 100 bronchograms			13.5	

The existence of bronchiectasis is proved bronchographically in 1 out of every 6 suspects. It is immediately apparent that the majority of the patients subjected to diagnostic study because of chronic cough do not have bronchiectasis. Bronchoscopy in these patients discloses chronic changes in the bronchial mucosa. Bronchograms fail to reveal bronchiectatic changes. A diagnosis of chronic bronchitis has been applied to such patients, such a diagnosis being arrived at by elimination. Compared with the incidence of other

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*Chief, Thoracic-Cardiovascular Surgery Service, Brooke Army Hospital, Fort Sam Houston, Texas.

**Chief, General Surgery Service, Letterman Army Hospital, San Francisco, Calif.

chronic pulmonary diseases, bronchiectasis has become a relative rarity thereby confirming the prediction that this disease entity would decrease in frequency as a direct result of more efficient management of acute respiratory infection in children and adults. More effective treatment has evolved concomitantly with the development of broad spectrum antibiotics.

CLINICAL MATERIAL

One hundred and fifteen consecutive proved cases of bronchiectasis were studied. The disease was unilateral in 95 patients and bilateral in 20 patients. One hundred and seven patients were treated by pulmonary resection—103 unilateral resections and 4 bilateral resections, totaling 111 operations. Eight of the 115 patients were not treated surgically. Eighty-two or 76.6 per cent were male; 33 or 23.4 per cent were female. There were only 5 Negro adults in the group. Six children are included in the series.

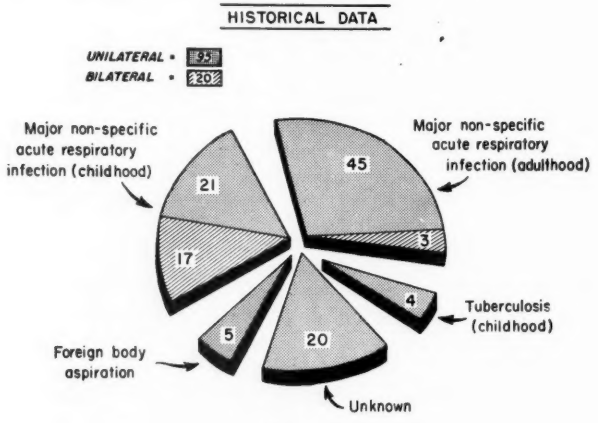

Fig. 1.

The symptomatology was consistent with that recorded in numerous reports and merits no repetition. Significant sputum volumes were recorded only in patients with severe bilateral disease. The ages range from 2 to 63, with an average age of 28. The average duration of symptoms was 6 years.

In 82.6 per cent of the patients studied, a positive significant history could be obtained. The childhood origin of the disease was consistently emphasized by repeated bouts of moderate upper respiratory infection during the winter months which tended to limit the school attendance. Since the report of Ochsner and associates[9] in 1949, little attention has been paid to the medical history of these patients. The fact that only 33 per cent of the patients studied were able to trace the origin of their disease to childhood suggests that the majority of cases no longer develop during childhood.

Of the patients studied, 41.7 per cent traced the onset of their bronchiectasis to a documented major respiratory infection in adulthood. This is significant as it establishes the fact that bronchiectasis can and does develop in

the adult. Three of the patients studied had bronchograms carried out early during their convalescence from atypical viral pneumonia. The initial films revealed bilateral basilar pseudo-bronchiectasis. Within a 6-month period, during which repeated bronchography was performed, bronchiectatic changes disappeared on one side and become permanent on the other. Resected specimens in each instance revealed the permanent pathologic changes of cylindrical bronchiectasis. It has been observed that the pseudo-bronchiectasis, which follows atypical pneumonia in the young adult, will clear usually within

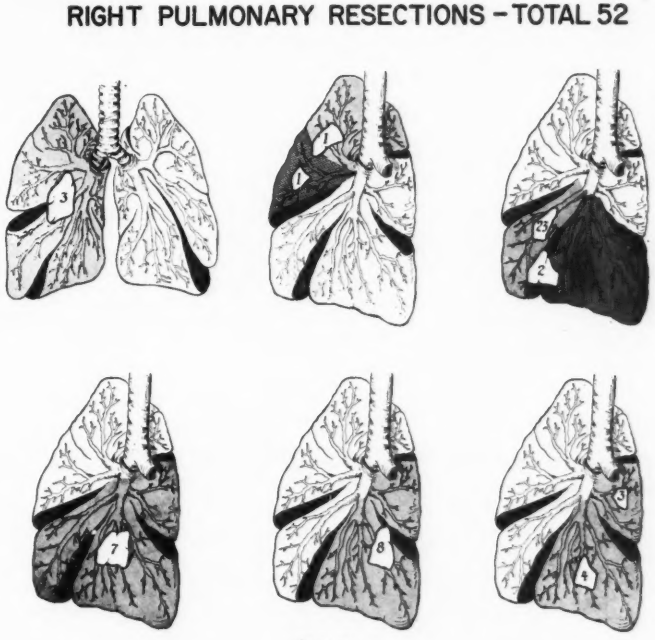

Fig. 2.

90 days following the disappearance of acute symptoms. Aggressive therapy of acute pneumonic processes in the adult must include adequate attention to airway toilet if the development of irreversible bronchiectasis is to be prevented.

The physician treating acute respiratory infections in the adult should be reminded of the statement by Gross[6]: "Bronchiectasis rises slowly out of the smoldering ashes of some previous pulmonary insult or long continued pulmonary inflammation."

PULMONARY RESECTIONS

Unilateral disease is almost equally distributed between the two lungs with the marked predilection for the lower lobes. Bilateral disease was present in 20 or 17.3 per cent of the patients studied. Of these, 60 per cent were treated with unilateral resection; 20 per cent had bilateral resections.

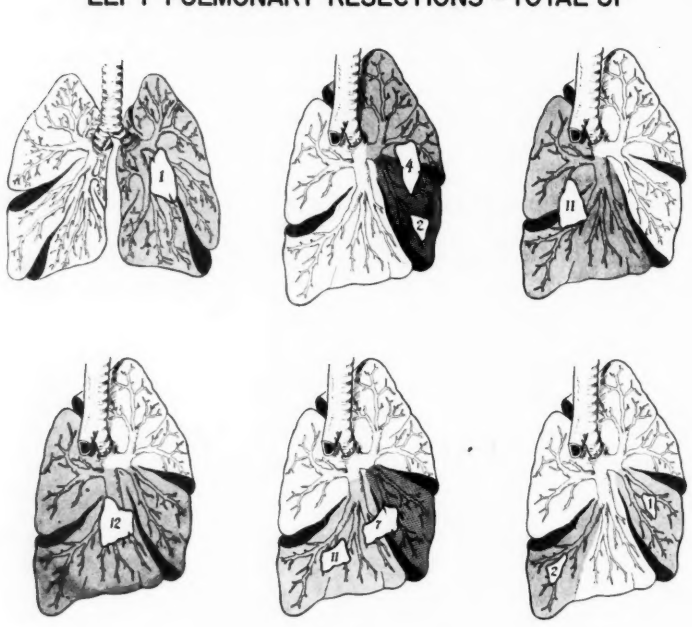
LEFT PULMONARY RESECTIONS - TOTAL 51
1
4
2
11
12
7
11
11
2

Fig. 3.

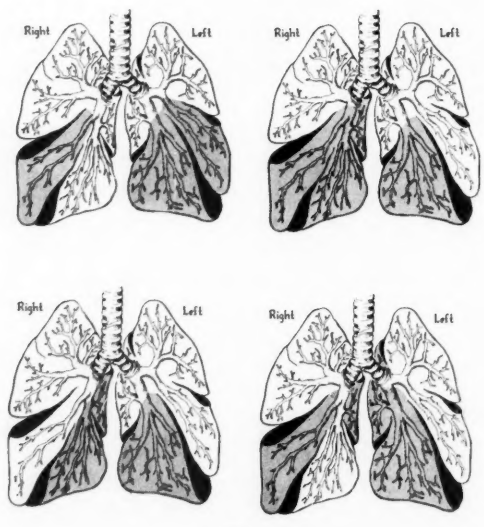
STAGED BILATERAL PULMONARY RESECTIONS - TOTAL 8
Right
Left
Right
Left
Right
Left
Right
Left

Fig. 4.

Eight patients in the series received no surgical treatment. Four had severe bilateral disease with critical maximum breathing capacities. Three with unilateral disease refused treatment. One patient had severe contralateral residual disease following previous lobectomy in another institution. There are no pulmonary cripples as a result of bilateral resection; the 4 patients enjoy reasonable health and are fully active.

In all operations, hilar dissection has been used with individual ligation of the vessels and closure of the bronchial stump with interrupted nonabsorbable sutures. In lower lobe resections it has been routine to preserve the superior segment when possible. This has not resulted in an increase in complications and its economic soundness is attractive. Preoperatively, patients have been carefully instructed in breathing exercises and this exercise is started under supervision the day after operation. Aggressive airway care, consisting of tracheal aspiration and coughing, is started on a 15 to 30 minute routine as soon as the patient reacts from anesthesia. Water-seal drainage of the pleural space is maintained for at least 48 hours and longer if complications occur. Pleural space suction was used only when a bronchopleural fistula developed. Penicillin and streptomycin have been administered for 7 days postoperatively. Preoperative antibiotics have been used only in bilateral disease.

COMPLICATIONS

A total complication rate of 19.8 per cent is considered high but not unreasonable when it is realized that 70 per cent of the operations were performed by residents in their third and fourth years of general surgical training. In view of complication rates of 25.8 per cent reported by Ginsberg[5] in 1955, one of 22.8 per cent reported by Bradshaw[1] in 1957, and 41.6 per cent reported by Helm[7] in 1958, it appears that a rate in the range of 20 per cent is acceptable with current techniques.

TABLE II. COMPLICATIONS (111 OPERATIONS)

TYPE	NUMBER	TREATMENT	RESULT
Bronchopleural fistula	8	Tube drainage - 7 Thoracoplasty - 1	Cured
Bronchopleural fistula-empyema	4	Tube drainage - 4	Cured
Hemothorax	3	Thoracotomy - 3	Cured
Empyema	1	Tube drainage - 1	Cured
Atelectasis	1	Bronchoscopy - 3 times	Cured
Hemoptysis	1	Bronchoscopy, conservative - 1	Cured
Minor wound infection	3	Local	Cured
Major wound infection	1	Secondary closure	Cured

Complication rate — 19.8%
Bronchopleural fistula — 54.5%
75% of bronchopleural fistulas associated with stump of lingula bronchus

As the direct result of constant awareness of the problems resulting from retained secretions, it has been possible to prevent complications directly

traceable to this factor. Each patient receives preoperative indoctrination as to the technique and value of coughing. Breathing exercises are taught preoperatively and resumed under supervision on the first postoperative day. Any patient who develops significant secretions during operation is bronchoscoped at the conclusion of operation. Tracheal suction is used on a 15 to 30 minute schedule during the first 48 hours. In the early postoperative period, bronchoscopy is performed if the efficiency of the tracheal suction or cough is questionable. Consequently, the development of atelectasis is rare. Such a program necessitates round the clock attention by the house staff.

Bronchopleural fistula associated with a segmental bronchial stump is a well-recognized hazard and is most likely related to the destruction of bronchial vessels incidental to the resection technique. Complete skeletonization of the segmental bronchus was frequently evident in the operations performed during the first few years of this experience, the period in which the major portion of the complications appeared. The only significant variation in technique has been the adoption of minimal dissection in the region of the origin of the segmental bronchus to produce a bleeding bronchial stump in preference to one which is devoid of peribronchial tissue. Following closure of the bronchial stump, the bleeding is controlled by superficially placed suture ligatures. It is our clinical impression that this has been significant in reducing the instance of bronchopleural fistula. We have not been aware of lung difficulty traceable to operative injury of intersegmental veins.

Intrapleural air pockets which appear following removal of the thoracotomy tubes are considered as diagnostic of fistula and are treated by suction drainage. The early use of tube drainage seems to reduce the incidence of empyema as a concomitant complication of fistula.

Complications have contributed to poor results in 2 of 4 patients not improved by operation. Of the complications listed, 60 per cent occurred during the first 5 years of the experience.

RESULTS

TABLE III. RESULTS

RESULTS	UNILATERAL DISEASE	BILATERAL DISEASE		FOLLOW-UP PERIOD	PER CENT	BENEFITED
		I	II			
Cured	27	1		3.7 yr.	36.4	94.8%
Improved*	37	4	4	5 yr.	58.4	
None	3	1		3 yr.	5.2	5.2%

*Two late deaths occurred in patients who were improved.

Of 107 patients treated by resection we have follow-up information on 77 or 71.9 per cent; of these 75 are alive. There have been two late deaths not attributable to bronchiectasis.* One patient died of carcinoma in the opposite lung 2 years after operation. One patient died of generalized peritonitis 3 years following lobectomy. Thirty patients have been lost to follow-up.

*These deaths were in patients who were improved.

"Cured" designates those patients who are free of symptoms and have no limitation of activity; 36.4 per cent of the group fall into this category. The patient with a single operation in bilateral disease has had no progression of mild anatomic changes in the unoperated lung.

"Improved" designates those patients who continue to have a cough but are able to carry out normal physical activity; 58.4 per cent of the group fall into this category. In addition to cough, minor occasional hemoptysis is reported by 5 patients. Increased susceptibility to respiratory infection is reported by 3 patients. Three of the 4 patients undergoing bilateral resection report mild dyspnea on exertion. It should be noted that the residual cough is frequently described as dry in distinction to the characteristic productive cough that existed prior to operation. A significant number of the patients who continue to cough have resumed smoking since operation.

"Poor" is applied to those patients who have not been improved by operation. Marked productive cough continues and their physical activity is limited to the point that they cannot be gainfully employed; 5.2 per cent fall into this category.

Seventy-three patients or 94.8 per cent were benefited by resection and are employed.

Comparison of Results.—A survey of the surgical experience with bronchiectasis published during the period of this study may be used as a basis for predicting results in surgical treatment of the disease. A complication rate of 22.8 per cent can be anticipated; however, the mortality rate should remain less than 1 per cent. An average cure rate of 43.2 per cent is expected while another 42.6 per cent should be improved by operation. Of those patients treated, 85.8 per cent should be benefited and capable of normal activity following operation. Surgical treatment will fail in approximately 6.2 per cent.

TABLE IV. COMPARISON OF RESULTS

AUTHOR	DATE	PATIENTS	% CURED	% IMPROVED	% POOR	% MORTALITY	% BENEFITED
Ochsner et al.[9]	1949	82	44.9	34.4	4.2	0	79.3
Ginsberg et al.[5]	1955	221	75.0	16.0	5.0	0	91.0
Bradshaw et al.[1]	1957	270	21.1	61.5	6.6	0	82.6
Helm and Thompson[7]	1958	159	41.0	43.0	10.0	6	84.0
Brooke Army Hospital	1959	77	36.4	58.4	5.2	0	94.8

DISCUSSION

The aggressive diagnostic approach generally utilized in the evaluation of patients with symptoms of pulmonary disease is essential in the diagnosis and management of bronchiectasis. That bronchiectasis can originate in the adult is becoming well established, a fact which will modify the earlier concept of the disease being one which generally originated during childhood.

Classification of bronchiectasis into primary and secondary types is of didactic interest only. A true bronchial obstructive phenomenon exists in the

early phases of the disease. There is no evidence to suggest that the pathologic changes, distal to the site of foreign body bronchial occlusion, differ in any way from those changes which develop distal to bronchial occlusion which is the result of localized edema and infiltration occurring in acute pulmonary infection. The combination of some degree of bronchial obstruction and distal inflammation is constant in the development of bronchiectasis.

The continued reporting of surgical experiences with bronchiectasis confirms the efficacy of resection as both a palliative and curative measure. The advanced saccular type disease appears to be decreasing in frequency, with the advanced cylindrical pattern being noted frequently. This is undoubtedly due to earlier use of bronchography in the evaluation of patients with pulmonary symptoms; thus the bronchial changes are recognized before they progress to marked saccule formation. Pulmonary resection is never recommended solely on the basis of radiographic changes but is reserved for those patients in whom there is an unquestionable history of chronic pulmonary symptoms associated with permanent changes in the bronchographic pattern.

The complication and mortality rates associated with earlier experiences of pulmonary resection for bronchiectasis have constantly decreased with the use of individual dissection and ligation of hilar structures, the general availability of endotracheal anesthesia, alert postoperative care aimed at maintaining a clear airway, and the prevention of infection by the use of antibiotics to counteract pleural contamination incidental to operation.

Pulmonary resection is the treatment of choice in all age groups.

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INFLUENZAL PNEUMONIA AND BLOOD GAS ANALYSIS

An Attempt at Correlation Between Anatomic and Functional Findings

Milorad Andrial, M.D., Manchester, Conn.

ONE of the striking findings in gas analyses of arterial blood in patients with influenzal pneumonia was a marked reduction of oxygen saturation levels.[1, 2] This fact was advanced to explain the deep cyanosis of the patients.

The cause of this insufficient oxygen saturation may be sought in the following factors: (1) obstruction of alveolar and bronchiolar lumen by secretion, edema fluid, blood, and exudate, (2) impairment of gas diffusion because of thickening of alveolar wall by edema and inflammatory exudate, and (3) hypoventilation caused by pleural pain and possible toxic damage to the respiratory center.

We have been mainly interested in examining the role played by the lung arteries and arterioles in this process. According to von Euler and Liljestrand,[3] inspiration of air poor in oxygen or rich in carbon dioxide results in a rise of pulmonary pressure in the animal. If such arterial contraction would come into play to diminish the blood supply to individual, poorly aerated, lung portions, then—according to these authors—this would be a protective mechanism; among other things, little or no mixing of nonoxygenated blood from alveolar capillaries (e.g., because of diffusion impairment due to alveolar exudate) with blood rich in oxygen from healthy lung portions would occur.

This mechanism would allow blood that originally was to flow into poorly aerated lung areas to be deviated into healthy lung tissue. This would maintain the same level of blood pressure in the pulmonary artery, provided of course that sufficient healthy lung parenchyma were available as reserve.

Animal experiments and investigations in human beings by Motley and his associates,[4] who introduced air with a low oxygen content into one and subsequently both lungs, seem to confirm this point of view, inasmuch as no blood pressure rise in the main pulmonary artery resulted from one-sided respiration.

What are the alterations of blood supply that occur in the lung of influenzal pneumonia? Clinically, it was seen that frequently only a small rise in oxygen

From the Institute of Pathology and the Bürgerspital, University of Basel, Switzerland.
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*Present address: Department of Pathology, Manchester Memorial Hospital, Manchester, Conn.

saturation of arterial blood resulted from oxygen breathing (Herzog[2]), and normal values of 95 to 97 per cent could not be obtained. Three main reasons could account for this: (1) arterioles situated in the pneumonic areas would not contract sufficiently to prevent mixing of blood poor in oxygen with blood rich in oxygen; (2) the pneumonic process was too generalized and there were no reserves of healthy functioning lung parenchyma where oxygenation could take place; and (3) unsatisfactory respiratory movements. Naturally also combinations of the three factors would be possible.

We have tried to find out—by studying the postmortem material in 3 typical cases of influenzal pneumonia and comparing the findings with the results of blood gas analyses—whether or not there was a contraction of arteries in the pneumonic areas. We examined by serial sections the material from pneumonic areas: in Case 1, 68 sections; in Case 2, 24 sections; and, in Case 3, 54 sections. In order to make the arteries stand out better we used, in addition to hematoxylin and eosin stain, Weigert's fuchsin elastic tissue stain.

CASE REPORTS

CASE 1.—G. G., a 55-year-old man, had never been seriously ill before. On Oct. 14, 1957, fatigue set in; the following day noisy respiration was noticed. On October 16, the temperature rose to 38.5° C. He was admitted to the hospital on October 18. Loss of weight and dyspnea were apparent; râles were heard over both lung fields, and a friction rub over the right base was present. He was treated with penicillin, erythromycin, streptomycin, hydrocortisone, blood transfusions, and oxygen therapy. On October 19 (the sixth day of illness and 2 days before death), blood gas determination of arterial blood was performed [italics are ours].

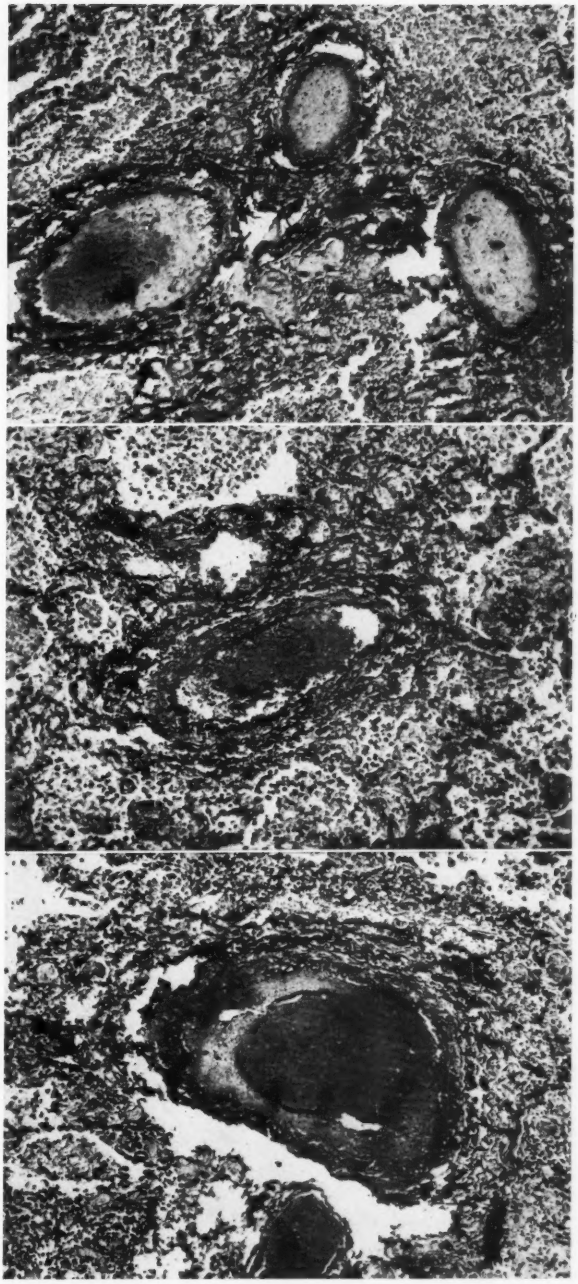
	Normal	Patient
O_2 capacity (vol. %)	19.5-20.5	20.7
O_2 saturation (%)	95-97	*38.1*
CO_2 content (vol. %) (plasma)	54-57	66.8
pH (plasma)	7.38-7.41	7.38
CO_2 tension (mm. Hg)	40.0	50.5

Among the results, the severe arterial hypoxemia stood out, and a massive functional shunt was suspected. The patient died on October 21, the eighth day of illness, vomiting coffee-ground-like material.

The autopsy report (S 1337-57, Professor Werthemann) [italics are ours] stated that there were severe confluent bronchopneumonia and interstitial pneumonia of *both lower lobes and the left upper lobe,* also isolated bronchopneumonic foci of the *right middle and upper lobes,* total obliteration of both pleural cavities with fresh fibrinous pleuritis of the right lower lobe; severe, in part hemorrhagic tracheobronchitis. Fresh mucosal ulcerations of the first duodenal portion were seen also.

The pathologic findings showed that all lung lobes were more or less involved by the pneumonic process.

Sixty-eight microscopic sections taken from the diseased lung portions showed an almost complete obliteration of alveoli and bronchioles by inflammatory exudate, and marked dilatation and congestion of all blood vessels, i.e., capillaries, small and large veins, and arteries (Fig. 1, *A, B,* and *C*). Of particular interest were the markedly distended capillaries in the alveolar wall.



A.

B.

C.

All sections were stained with hematoxylin and eosin and Weigert's fuchsin elastic tissue stains.

Fig. 1.—Case 1. ***A,*** **Three markedly distended and congested arteries with numerous capillaries. Alveoli are filled with exudate containing abundant numbers of neutrophils with many histiocytes and erythrocytes. (×70; reduced ⅙.)**

B **and** ***C,*** **Similar histologic picture as in** ***A.*** **(×100; reduced ⅙.)**

Summary.—A 55-year-old man became ill suddenly and died 8 days later with generalized bronchopneumonia. Blood gas analysis performed 2 days before death showed a markedly reduced oxygen saturation of arterial blood. Detailed microscopic examination of the diseased lung portions showed, in addition to massive pneumonia, severe dilatation and congestion of lung arteries and arterioles.

CASE 2.—A. S., a 22-year-old man, had never been seriously ill. On Nov. 16, 1957, cold and cough, without expectoration, and slight fever developed. In the following days, increase of temperature, tachypnea and chest pain with respiratory movements occurred. On November 18, he was admitted to the hospital in poor general condition, with cyanosis of lips and tongue. Respiration was superficial, with tachypnea, and decreased respiratory sounds on both sides. The temperature was 40° C. X-ray studies of the chest showed bronchopneumonic infiltrates in the right upper lobe and left upper lobe.

Blood gas analysis of arterial blood on November 18 (on the third day of illness and 1 day before death) follows [italics are ours].

	Normal	10 min. without oxygen	20 min. with oxygen
O_2 capacity (vol. %)	19.5-20.5	20.6	20.7
O_2 saturation (%)	95-97	*81.2*	87.5
O_2 tension (mm. Hg)	85-95	43.0	—
CO_2 content (vol. %) (plasma)	54-57	48.5	47.1
pH (plasma)	7.38-7.41	7.49	7.50
CO_2 tension (mm. Hg)	40.0	29.5	28.0

The examination showed, among other findings, a reduced oxygen saturation of arterial blood that increased only slightly following oxygen breathing. It was considered likely that a functional vascular shunt and impaired alveolocapillary diffusion of oxygen had taken place.

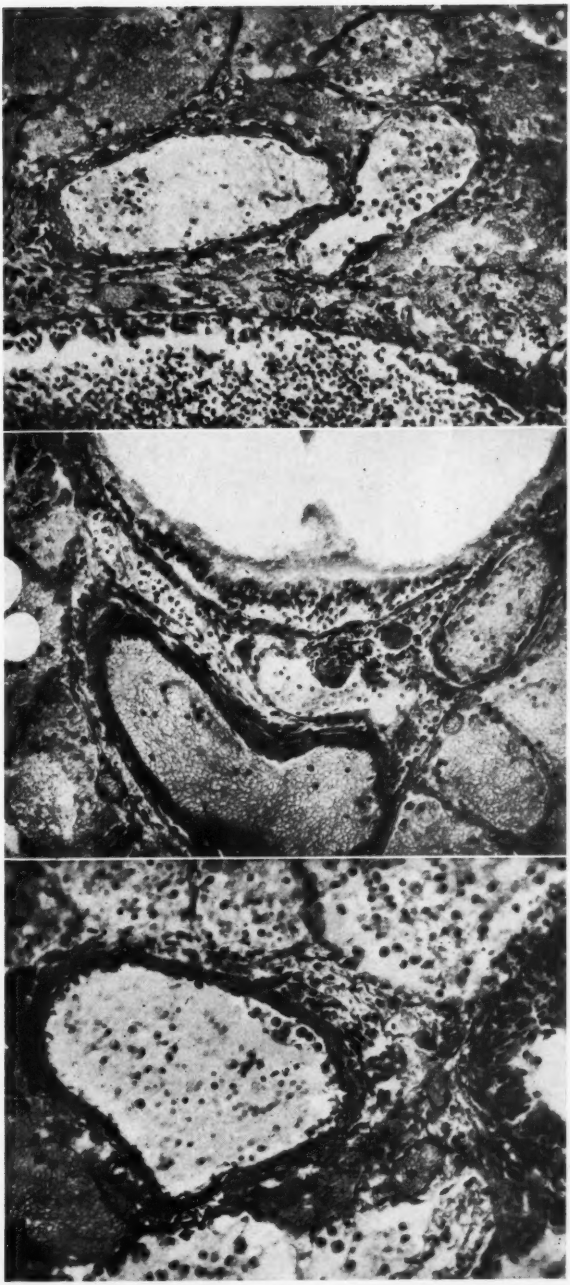
The patient was treated with penicillin, streptomycin, hydrocortisone, and analeptic drugs. The blood pressure kept falling and the patient died in cardiovascular collapse on November 19, the fourth day of illness.

The autopsy report (S 1463-57 by Professor Werthemann) [italics are ours] stated that there were severe confluent, hemorrhagic, in part suppurative bronchopneumonia of the *entire left lower lobe, the right upper and lower lobes,* fibrinous pleuritis overlying the pneumonic lung areas, terminal pulmonary edema, severe pseudomembranous hemorrhagic bronchitis and tracheitis.

Virological examination of the involved lung revealed the presence of influenzal virus (A asiatica). Bacteriologic examination of the lung showed *Staphylococcus pyogenes* var. *aureus.*

Microscopic examination of the lung (24 sections) displayed a massive obstruction of the alveolar and bronchiolar lumen by pneumonic exudate and a generalized severe congestion and dilatation of all blood vessels, i.e., also of the arterioles and capillaries. The picture was strikingly similar to that seen in the preceding case (Fig. 2, *A, B, C, D, E*).

Summary.—A previously healthy young man became suddenly ill with influenza and died after a course of only 4 days, with bronchopneumonia of



A.

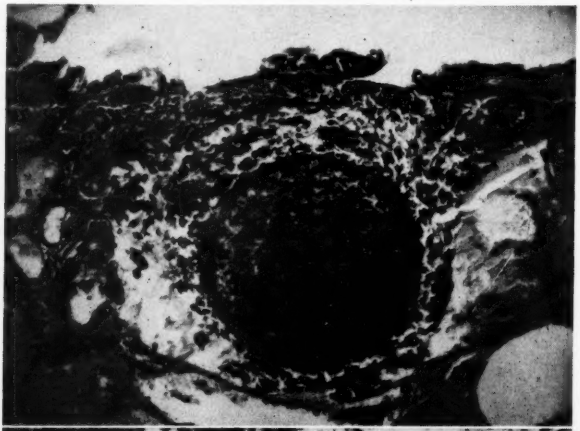
B.

C.

Fig. 2.—Case 2. *A*, *B*, *C*, *D*, and *E*, Marked dilatation of artery and vein. Bronchus contains numerous neutrophils, epithelium is in part desquamated. Alveoli are also distended and filled with blood. Note marked engorgement of interalveolar capillaries. (All magnified ×175, except *C* which is ×270; all reduced ⅙.)

the left lower lobe and the right upper and lower lobes. Blood gas analysis performed 1 day prior to death showed a diminished oxygen saturation of arterial blood which rose but slightly following oxygen breathing and did not reach normal values. Microscopic examination of the diseased pulmonary vessels revealed, besides severe pneumonic infiltrations, a generalized congestion and dilatation of lung arterioles.

D.



E.

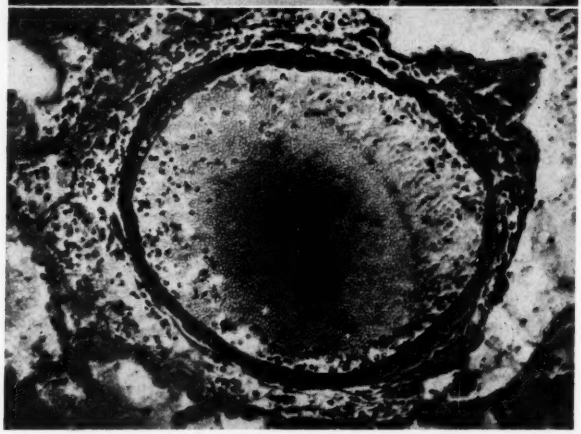


Fig. 2.—Cont'd. *D* and *E, For legend see preceding page.*

CASE 3.—J. M., a 71-year-old man, a former blacksmith, had been hospitalized a year previously for cirrhosis of the liver. He became ill on Nov. 18, 1957, with a fever of 39° C. and cough. On November 21, cyanosis of the lips was observed. Hospital admission occurred on November 22; cyanosis of the tongue was then also noticed. Blood pressure was 220/100 mm. Hg. X-ray studies of the chest showed pneumonic infiltrates of both lower lobes and possibly also of the right middle lobe. The patient was treated with penicillin, strophantin, and analeptic drugs.

On November 23, the sixth day of illness and 1 day prior to death, gas analysis of arterial blood was performed [italics are ours].

	Normal	10 min. without oxygen	20 min. with oxygen
O_2 capacity (vol. %)	19.5-20.5	19.0	19.0
O_2 saturation (%)	95-97	*83.0*	*89.5*
O_2 tension (mm. Hg)	85-95	50.4	61.5
CO_2 content (vol. %) (plasma)	54-57	41.0	41.0
pH (plasma)	7.38-7.41	7.38	7.37
CO_2 tension (mm. Hg)	40.0	31.0	31.5

The examination showed among other findings a reduced oxygen saturation of arterial blood that rose slightly following oxygen breathing but did not attain normal values. In spite of oxygen therapy and chloromycetin, the patient become somnolent and died on November 24 with the signs of progressive cardiac insufficiency.

The autopsy report (S 1490-57, Professor Werthemann) [italics are ours] stated that there were severe hemorrhagic, in part fibrinous tracheobronchitis, a lobar, confluent hemorrhagic pneumonia of the *right lower lobe;* confluent nodular hemorrhagic, purulent bronchopneumonia of the *left lower lobe;*

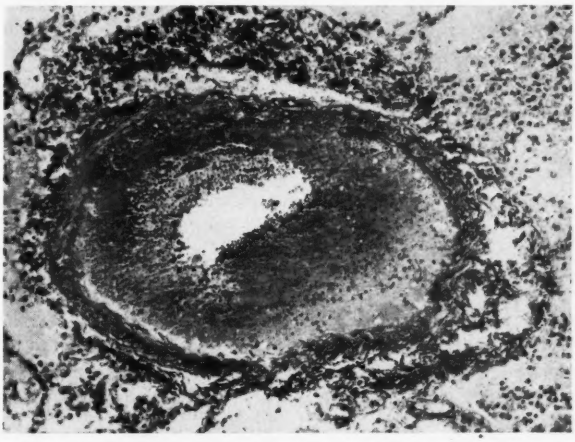


Fig. 3.—Case 3. Marked dilatation and congestion of an artery. Alveoli are filled with exudate consisting of neutrophils and erythrocytes. Deposition of anthracotic pigment. (×110; reduced ⅙.)

moderate edema and severe cyanosis of both lungs. There were also obliteration of the right pleural cavity, hypertrophic cirrhosis of the liver, and hypertrophy of the left ventricle and stenosing coronary sclerosis.

Microscopic examination of the diseased lung portions (54 sections) showed —similar to the previous cases—an almost complete obstruction of the alveolar and bronchial lumen by exudate, blood, and edema fluid and a generalized severe dilatation and congestion of all blood vessels, including lung arterioles (Fig. 3).

Summary.—A 71-year-old man with cirrhosis of the liver became ill suddenly with influenza and died 7 days later with lobar pneumonia of the right lower lobe and bronchopneumonia of the left lower lobe. Blood gas analysis performed 1 day before death displayed a reduced oxygen saturation of arterial blood that rose little following oxygen breathing but did not reach the normal

value. Microscopic examination of the diseased lung portions showed obliteration of alveolar and bronchiolar lumen by exudate and edema fluid and generalized congestion and dilatation of all blood vessels.

DISCUSSION

The microscopic picture of influenzal pneumonia is characterized in all the cases by a massive infiltration and obstruction of the respiratory passages by exudate with many polymorphonuclear leukocytes, fibrin, many erythrocytes, and edema fluid. The blood vessels are engorged with blood and are markedly distended.

It is a well-known fact that an acute inflammation of vascular organs is followed by dilatation and congestion of local blood vessels as shown clinically by the signs of hyperemia and swelling. The cardinal symptoms of Celsus, i.e., rubor, tumor, calor, and dolor, point to this fact. In an acute inflammatory process in the lung this organ does not act otherwise and hyperemic congestion and dilatation are easy to see. However, these are autopsy findings in a full-blown inflammation and it is not altogether admissible to draw conclusions from these findings regarding vascular reaction in the *early* stages of the illness.

In the present investigation we have been mainly interested in correlating the clinical findings made in the light of blood gas analyses and the pathologic findings after death. The cases just described have several things in common: the stormy course of the illness that takes them all ad exitum within 8 days in spite of their various ages (22, 55, and 71 years, respectively,) and the very similar pathologic findings of influenzal pneumonia. The blood gas analyses were performed in every case 1 or 2 days before death.

When examining the oxygen saturation rates of arterial blood, one is struck by the unusually low value in Case 1–38.1 per cent (normal value 95 to 97 per cent). It is likely that the generalized pneumonia involving all lung lobes was instrumental in reducing the oxygen saturation to such an abnormal level.

In Cases 2 and 3, the oxygen saturation levels were similar, i.e., 81.2 per cent and 83.0 per cent, respectively. This saturation was slightly increased further by breathing pure oxygen. In these 2 cases not all of the lung portions were involved by pneumonic infiltrates as it had been in Case 1, and this may explain in part the different findings in these two groups of cases.

A question that remains unanswered in the present investigation is whether pulmonary arterioles contract in the *early* phases of influenzal pneumonia and thus possibly prevent a functional shunt, an arteriovenous anastomosis.

The experiments of von Euler and Liljestrand[3, 5, 6] who administered air poor in oxygen or rich in carbon dioxide were carried out on animals and human beings whose lungs very likely were not subject to an acute inflammatory process. It is therefore quite possible that lung arterioles in man react to oxygen lack by contracting and that, in such a way, blood is channeled into better aerated lung areas. However, if a massive acute inflammatory process takes place, such as influenzal pneumonia, this mechanism of purposeful blood distribution is probably abolished, the arterioles dilate in order to accommodate

the increased blood volume attracted by the inflammatory reaction. A vascular reaction similar to that seen in influenza is equally observed in other types of acute pneumonia. Fig. 4 shows a microphotograph of a case of noninfluenzal bronchopneumonia. Like in influenza, an obvious dilatation and congestion of arteries and veins surrounded by inflammatory exudate in the alveoli and bronchioles is displayed.

Occasionally, blood supply to the diseased lung will be diminished not by arterial contraction but as a result of vascular thrombosis. Now and then we have seen such thrombotic occlusions in influenzal pneumonia especially in the chronic cases. These thromboses are—of course solely from the standpoint of gas exchange in the lung—useful and cause the blood stream to be diverted to noninvolved lung areas.

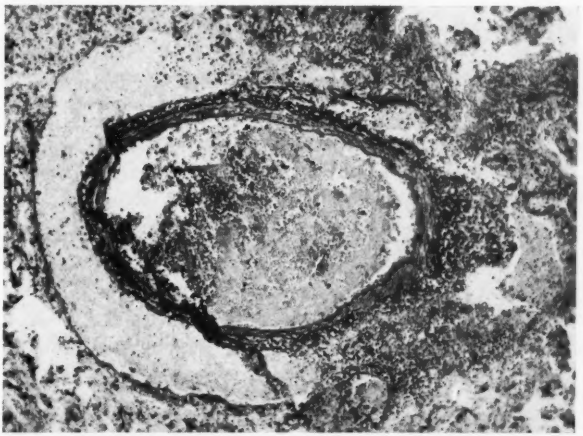


Fig. 4.—Nongrippal pneumonia. Dilated and congested artery and vein. Alveoli filled with exudate. (× 100; reduced ⅕.)

Remarks Regarding Oxygen Therapy of Patients Ill With Influenzal Pneumonia.—If a blood analysis shows that oxygen saturation of arterial blood is lower than normal, and if oxygen breathing does not raise it to normal values or does not affect it at all, it may be assumed that the gas exchange in the lung has been considerably impaired provided, however, that this did not occur as a result of lowered hemoglobin-oxygen binding capacity as for example in anemia (this would be characterized by a diminished oxygen capacity of arterial blood) or of poor respiratory movements, failure of the heart pump or of the respiratory center.

Oxygen respiration is frequently a lifesaving measure in many of the above conditions but fails if for anatomic reasons it is not possible to bring about a gas exchange in the lungs; an example of such a condition can be found in the acute influenzal pneumonia as we have shown it in three examples. Here the extent of inflammation is of course all important. If, as in Case 1, the inflammatory reaction almost completely involves both lungs, it becomes difficult to

hope that the oxygen level of arterial blood will rise significantly or even reach normal values as a result of oxygen breathing, since the oxygen is not able to penetrate into alveolar capillaries because of a mechanical blocking of the alveoli.

A more logical therapeutic approach to these conditions, characterized by a low arterial oxygen saturation not responding to oxygen breathing, would consist in oxygenizing blood directly instead of trying to have oxygen cross the blocked alveolo-capillary barrier in order to combine with the hemoglobin of capillary blood.

Already in 1882, von Schröder[7] had tried to oxygenize venous blood flowing through isolated organs. A modern method of oxygenization of venous blood was first suggested by Lillehei, DeWall and co-workers.[8, 9] Its use in operations on the heart and lungs is well established today. Oxygenization of venous blood is carried out in an oxygenator in which oxygen is mixed with venous blood in a special chamber; another important component of this machine is a debubbling device serving to remove the numerous gas bubbles from oxygenized blood to prevent gas embolism.

The main field of application of these oxygenators is naturally in heart surgery where they replace the heart and lung functions during the surgical intervention. In the present cases the problem consists in replacing only the function of the lung in the gas exchange, i.e., in the oxygenization of venous blood.

The technique of an artificial oxygenization of venous blood will have to be tried out, of course, in animal experiments. The duration of oxygen transfusion is limited to a large degree by hemolysis that occurs during such a procedure; moreover, changes in pH and electrolytes of blood occur and have to be corrected. Furthermore, one of the main technical problems of an oxygenator is still a quantitative prevention of gas bubble formation and gas embolism.

If these technical problems could be reduced and eliminated then, in the uncomplicated case, blood could be removed from a vein and run through an oxygenator; the oxygenized blood would then be transfused into another vein from which it would reach the heart and flow through the diseased lungs, circulate in the arteries, and again return to the vein going to the oxygenator. It seems that such a procedure, if the technical problems could be mastered, might not be much more complicated than an ordinary blood transfusion.

The above remarks represents merely a therapeutical suggestion that we are advancing very humbly being aware of the technical problems involved. However, if it were possible to carry out such an artificial oxygenization of venous blood with a simple oxygenator, for a significant length of time without the risk of gas embolism, hemolysis, significant changes in the level of plasma ion concentrations, and the pH of blood, then we would have a new important therapeutical tool with the help of which the 3 patients just reported might have been saved.

Obviously, the method of artificial oxygenization of blood could be applied in many other conditions characterized by a partial or complete loss of lung function, such as generalized lung edema, lungs in drowning, trauma of the thorax, and traumatic lung injuries.

SUMMARY

An attempt is being made to correlate the low oxygen saturation levels of arterial blood in 3 patients dying of influenzal pneumonia with the microscopic findings in their lungs. No vascular contraction—as expected on the basis of experiments done by von Euler and Liljestrand—was seen in the diseased lung areas.

In cases where oxygen breathing is followed by no rise or only a small rise of oxygen saturation levels in the arterial blood, artificial oxygenation of venous blood by means of an oxygenator is suggested as the logical treatment.

I wish to thank Professor Werthemann for allowing me to study autopsy protocols and autopsy material relative to this study, Professor Staub for permission to study and use parts of the case histories, and Dr. Herzog for suggestions regarding blood gas analysis. Microphotographs were made by Mr. Sutter.

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BILATERAL INFERIOR DEEP CERVICAL (SCALENE FAT PAD) AND PARATRACHEAL LYMPH NODE BIOPSIES

Alfred H. F. Lui, M.D., Wayne W. Glas, M.D.,* and*
Eugene H. Lansing, M.D., Eloise, Mich.

BIOPSY of the inferior deep cervical lymph nodes, as advocated by Daniels[1] in 1949, has proved to be a worthwhile procedure in the diagnosis of intrathoracic diseases. This operation is usually performed on one side and consists of a partial excision of the scalene fat pad with its associated inferior deep cervical lymph nodes. Steele and Marable[2] have described an excellent method of removing the paratracheal and inferior deep cervical lymph nodes on one side through an incision along the anterior border of the sternocleidomastoideus muscle. We have extended this procedure and at present it is our policy to perform bilateral inferior deep cervical and paratracheal lymph node biopsies through a single incision.

TECHNIQUE

The patient is placed in the supine position and the neck is extended by placing a folded sheet beneath the shoulders. With the surgeon on the left side, a transverse incision is made about 3 cm. above the suprasternal notch (Fig. 1, *1*). The ends of this incision should be located slightly lateral to the anterior border of each sternocleidomastoideus muscle. The incision is extended through the platysma muscle and the upper and lower flaps are undermined in the subplatysmal plane. The investing layer of the deep cervical fascia is incised vertically in the midline avoiding the anterior jugular veins and the jugular venous arch (Fig. 1, *2*). The sternohyoid and sternothyroid muscles are retracted laterally to expose the pretracheal layer of the deep cervical fascia. This fascia is incised vertically in the midline avoiding the underlying inferior thyroid veins and exposing the trachea in the superior mediastinum (Fig. 1, *3*). Lymph nodes with their accompanying adipose tissue from the right and left paratracheal areas are dissected free and removed.

The right scalene fat pad with its associated inferior deep cervical lymph nodes is partly excised by removing its medial and inferior portions which are adjacent to the internal jugular and subclavian veins. This is accomplished by retracting the sternocleidomastoideus muscle anteriorly and laterally after the

From the Department of Surgery, Wayne County General Hospital, Eloise, Mich.
Received for publication Oct. 12, 1959.
*Instructor in Surgery, University of Michigan School of Medicine.

investing layer of the deep cervical fascia along its anterior border has been incised for a short distance. The sternohyoid and sternothyroid muscles are retracted medially. The pretracheal fascia, forming the anterior portion of the carotid sheath, is incised longitudinally exposing the internal jugular vein which is retracted medially (Fig. 1, *4*). The scalene fat pad is palpated anterior to the scalenus anticus muscle in the subclavian triangle. The scalene fat pad is dissected off the prevertebral fascia covering the scalenus anticus muscle thus

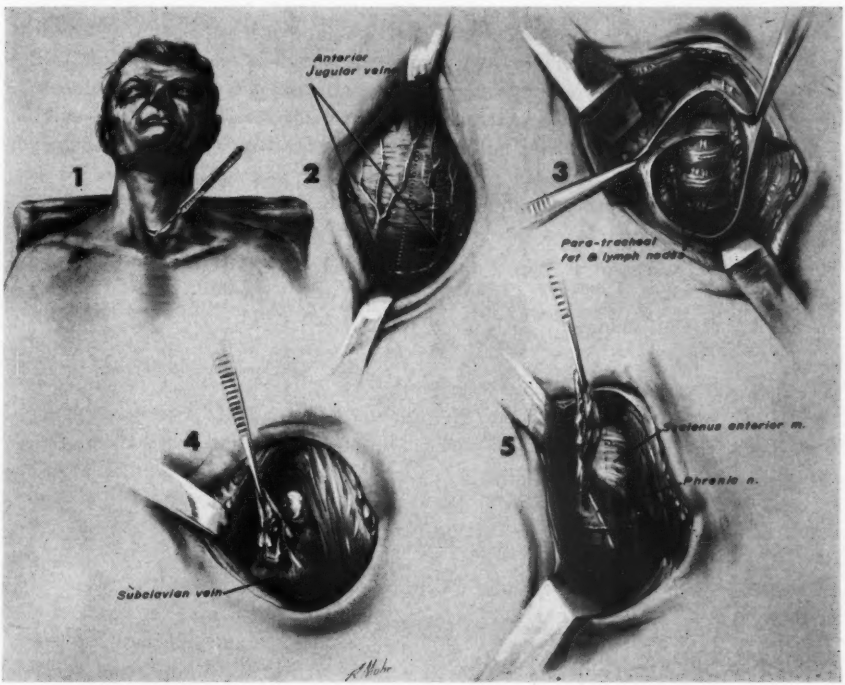

Fig. 1.—*1*, Transverse skin incision for bilateral inferior deep cervical and paratracheal lymph node biopsies.

2, Vertical midline incision through the investing layer of the deep cervical fascia.

3, Vertical midline incision through the pretracheal fascia exposing the trachea.

4, Exposure of the right scalene fat pad obtained by retracting the sternocleidomastoideus muscle anteriorly and laterally, and the internal jugular vein medially.

5, Exposure of the right scalenus anticus muscle with its overlying phrenic nerve after the scalene fat pad has been removed.

exposing the phrenic nerve (Fig. 1, *5*). It is also dissected off the proximal portions of the internal jugular and subclavian veins. The inferiomedial portion of the scalene fat pad with its associated inferior deep cervical lymph nodes is then excised.

The surgeon now moves to the right side of the table and the left scalene fat pad is removed in a similar manner. Particular care is exercised to prevent injury to the thoracic duct. Lymph nodes from the four different areas of biopsy are placed in separate specimen jars. After complete hemostasis is obtained

in all four areas of biopsy, the investing layer of the deep cervical fascia is approximated in the midline by interrupted sutures. The skin flaps, including the platysma muscle, are approximated with one layer of interrupted sutures placed so that the knots are buried in the subplatysmal plane. The superficial portion of these sutures is located in a subcuticular position and no skin sutures are used. A pressure dressing completes the procedure.

SUMMARY

A method of biopsy in which both the right and left inferior deep cervical and paratracheal lymph nodes are removed through a single incision is described. This eliminates a second biopsy through a separate incision on the opposite side. We believe that a greater number of positive diagnoses will be made when both right and left inferior deep cervical and paratracheal lymph nodes are biopsied routinely.

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SURGICAL RESECTION IN PULMONARY TUBERCULOSIS DUE TO ATYPICAL MYCOBACTERIUM TUBERCULOSIS

Raymond F. Corpe, M.D., and Joseph Liang, M.D.,** Rome, Ga.*

EXPERIENCE with over 180 cases of atypical pulmonary tuberculosis, treated with conventional antituberculosis drug therapy at Battey State Hospital, has shown the conversion rate to be about 22 per cent. In typical tuberculosis, the conversion rate is about 90 per cent after 6 months of drug therapy. In atypical tuberculosis, the great shortcomings of this medical approach have led us to continue excisional surgery in selected cases. Our experience would indicate that resection can be done with a low morbidity and a reasonably low mortality with resultant high sputum conversion in well-chosen cases and affords reasonably good results in the long-term tenure.

MATERIALS AND METHODS

From Jan. 1, 1950, to the end of April, 1959, 25 patients with atypical tuberculosis were subjected to pulmonary resection. Of the 23 white patients, 18 were male and 5 were female. The 2 Negro patients were male. Twelve patients subjected to resection were in their forties; 6 were in their thirties; 5 were in their fifties; and the youngest patient was 28 and the oldest 67 years of age.

According to the classification of the National Tuberculosis Association, the disease was minimal in 1 case, moderately advanced in 13, and far advanced in the remaining 10 cases.

On admission bacteriologic studies, 16 of the patients were positive by both direct smear and culture and 9 were negative on direct smear but positive on culture. Using Runyon's classification,[1] 22 of the positive cultures were nonphotochromogenics (Group III-Battey type). In 2 patients, there were photochromogenic strains (Group I), and in 1 there was a scotochromogenic strain (Group II).

As soon as a working diagnosis of pulmonary tuberculosis was established, 24 of the 25 patients were placed on antituberculous drug therapy. Twelve of these patients received isonicotinic acid hydrazide (INH) and para-aminosalicylic acid (PAS), 9 received INH, streptomycin, and PAS, 3 received streptomycin and PAS, and 1 patient received INH, streptomycin, PAS, and Dalicin

From the Battey State Hospital, Rome, Ga.
Received for publication Oct. 19, 1959.
*Superintendent.
**Chief of Surgery.

in the preoperative period. Additional chemotherapy was used in the postoperative period. Ten of the 12 patients having had INH and PAS received streptomycin in the postoperative period. Another patient who received INH and PAS had pyrazinamide (PZA) added in the postsurgical period. There was 1 patient who received no drug therapy before or after surgery.

Two patients had no sensitivity studies done prior to surgery. Of the 23 patients who did have, 23 were completely resistant to isoniazid, and 13 of these 23 were totally resistant to streptomycin–5 were partially sensitive, and 5 were completely sensitive to streptomycin on the last studies done prior to surgery.

Four of the 25 patients had negative sputum studies at the time of surgery; 1 patient had been negative for 3 months, 2 for 5 months, and 1 for 10 months. Of the remaining 21 patients, 2 were intermittently positive and 19 had never had negative cultures.

Twenty-three of these patients had preoperative bronchoscopies. One had a partial stenosis of the right upper lobe bronchus. One patient had severe endobronchitis which required postponement of surgery for 2 months until the inflammatory process had subsided under treatment. Resection was then successfully performed. Five of these patients had bronchograms. One revealed extensive bronchiectasis of the left lung and this patient eventually had a left pneumonectomy.

Twenty of the 25 patients were operated upon for open cavities plus positive sputum. One patient had an open cavity and a negative sputum, 1 patient had residual fibrocaseous disease and a positive sputum, 3 patients had residual fibrocaseous disease with a negative sputum.

There were 26 resections performed on these 25 patients (Table I). One patient had a right upper lobectomy first followed 20 days later by a segmentectomy by the removal of the upper division of the left upper lobe.

TABLE I. SURGICAL PROCEDURES

PROCEDURES	NO.	WITHOUT COMPLICATION	WITH COMPLICATION	DEATH
Subsegmentectomy	4	3	1	0
Segmentectomy	7	6	1	0
Lobectomy, alone	10	9	1*	1
Lobectomy, plus	3	1	2*	2
Pneumonectomy	2	2	0	0
Total	26	21	5	3

*In these cases, the complications were followed by death.

MORBIDITY AND MORTALITY

The patient who had bilateral resections had an uncomplicated course following each resection. Five patients (20 per cent) had postoperative complications (Table II). One of the 4 subsegmental resections was followed by a bronchopleural fistula which required post-resection thoracoplasty. One of the 7 segmental resections was also complicated by a bronchopleural fistula and residual space problem that required a post-resection thoracoplasty. There were no deaths in the 11 patients who had small resections.

TABLE II. MORBIDITY AND MORTALITY DUE TO SURGICAL COMPLICATION

PROCEDURES	COMPLICATION	MORTALITY	POSTOP. SPUTUM STATUS
1. Subsegmentectomy, apical posterior segment, LUL, with post-resection thoracoplasty	Bronchopleural fistula and residual space		Sputum converted 28 months after surgery
2. Segmentectomy, apical posterior segment, RUL, with post-resection thoracoplasty	Bronchopleural fistula and residual space		Sputum converted following surgery
3. RUL lobectomy plus segmentectomy, superior segment, RLL	Pneumonia	Died of pneumonia on seventh postop. day	
4. RUL and RML lobectomies plus wedge of superior segment, RLL, with post-resection thoracoplasty	Bronchopleural fistula and residual space	Died of pulmonary insufficiency on fourth postop. day	
5. RUL lobectomy	Pulmonary insufficiency	Died of hypoxia, 48 hours	
Total		5 Cases	

Of the 10 patients who had lobectomy only, 9 had no complications. One patient died of pulmonary insufficiency 48 hours after a right upper lobectomy. There were 3 patients who had lobectomies plus removal of other lung tissue, and 2 of these procedures were accompanied by complications which led to death. One was a 67-year-old white woman who had a right upper and a right middle lobectomy plus removal of a wedge from the lower lobe. There was not enough tissue to adequately fill the pleural space and a post-resection thoracoplasty was performed 3 weeks after the resection. Following this, the patient died of pulmonary insufficiency on the fifth postoperative day. Another patient, having had a right upper lobectomy plus a superior segmental resection of the superior division of the right lower lobe, died on the seventh postoperative day of pneumonia and atelectasis on the operative side, which was revealed at autopsy. There were 2 patients who had pneumonectomies and both had uncomplicated courses postoperatively. The surgical mortality rate, therefore, was 12 per cent.

Pathologic and bacteriologic studies of the surgical specimens revealed 2 surgical specimens negative on direct smear and culture. Twenty-four of the 26 surgical specimens had positive direct smears and, of these, 18 were positive on culture. According to C. Edwin Smith, Battey Laboratory Director, these cultures were the same in each case as had been isolated preoperatively from each individual. Dr. I. Stergus, Pathologist at Battey State Hospital, says that "Pathologically, these lesions are indistinguishable from those of typical tuberculosis, grossly and microscopically."

RESULTS

Four of the 25 patients had negative sputum at the time of surgery which remained consistently negative following surgery. Of these 4 patients who had

negative sputum, 1 had an open cavity and the other 3 had residual fibrocaseous disease. Whether or not the sputum of these 4 patients would have remained negative without surgery cannot be stated, but we count these 4 patients as combined medical-surgical treatment successes.

The sputum was positive in 21 patients at the time of surgery. In 14 cases, it converted immediately following surgery and has remained negative. We consider these 14 successes primarily attributable to surgery. There were 3 more patients who had cavitary disease with positive sputum at the time of surgery which did not convert immediately following surgery. The sputum of 1 patient converted within 3 months with new chemotherapy and in the other 2 converted after longer periods of chemotherapy. These successes are also attributed to combined surgical-medical therapy. Our failures are limited to 3 patients who died as a result of their surgery. We believe that 22 of the 25 patients, or 88 per cent, who are now alive and with negative sputum, are treatment successes with the main credit being given to the addition of surgical therapy (Table III).

TABLE III. SPUTUM STATUS FOLLOWING SURGERY

	NO.	PER CENT
Negative before and after surgery	4	16
Positive prior to surgery and negative thereafter	14	66
Positive prior to surgery and after surgery but still converted subsequently	4	16
Positive prior to surgery and died as a result of surgery	3	12
Total	25	100

DISCUSSION

In judging surgical therapy, one has to weigh the risk of surgery against the risk the patient must take without this treatment. In the group of patients at Battey State Hospital with atypical tuberculosis, only 20 per cent have been treated successfully by chemotherapy alone. In this small group of 25 patients treated by surgery and chemotherapy, 88 per cent have been treated successfully. It is our belief that patients who are surgical candidates should be subjected to resection as soon as possible. We have not attempted to reach a target point of negativity in this group.

The surgical mortality rate was 12 per cent. Up to the present time, there have been 31 deaths in the 155 patients treated medically, for a mortality rate of 20 per cent. In this medically treated group, 8 patients have died of atypical pulmonary tuberculosis, 7 have died of cardiac disease, and 13 have died of pulmonary tuberculosis and cardiac failure.

In a previous publication,[2] we suggested that resections of less than a lobe were unsatisfactory. With a somewhat larger experience, we found a complication rate of 20 per cent in resections of less than a lobe, and there were no deaths in this group. Although bronchopleural fistulas are serious complications, they are amenable to post-resection thoracoplasties. We believe that smaller resections may be used wherever indicated.

The deaths in our group of patients followed lobectomy plus segmental resection and/or wedge resections. Two of these three deaths might have been averted. The patient who had had a right upper and right middle lobectomy plus a wedge resection of the superior segment of the right lower lobe could have been treated better by a pre-resection thoracoplasty and then completion of the thoracoplasty and tailoring of her chest to fit the remaining lung tissue at the time of the resection. The patient who died from pulmonary insufficiency following a right upper lobectomy might not have been subjected to surgery if a more detailed preoperative pulmonary function evaluation had been made.

SUMMARY

1. Twenty-two of 25 cases of atypical tuberculosis are alive and well following combined surgical-medical therapy.

2. The morbidity rate was 20 per cent. Three patients had bronchopleural fistulas, 2 of whom were successfully treated by post-resection thoracoplasty.

3. There were 3 postoperative deaths.

4. Early resection is advocated in suitable candidates.

5. Resections of less than a lobe are also advocated when indicated.

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THE DEVELOPMENT OF BRONCHOGENIC CARCINOMA IN PATIENTS WITH PULMONARY TUBERCULOSIS

Roger E. Campbell, M.D., and Felix A. Hughes, Jr., M.D.,
Memphis, Tenn.

THERE has been considerable discussion in the past concerning the simultaneous occurrence of pulmonary tuberculosis and bronchogenic carcinoma.[1-4] Recent interest in this problem reflects the presumption that the incidence of bronchogenic carcinoma may be higher in patients with pulmonary tuberculosis than in the general population. This led us to review and analyze the 11,000 cases of pulmonary tuberculosis and 650 cases of bronchogenic carcinoma seen at Kennedy Hospital between the years 1947 and 1958.

During this 10-year period, 24 patients with proved pulmonary tuberculosis and bronchogenic carcinoma were seen. Active pulmonary tuberculosis unquestionably existed prior to the onset of the carcinoma in 10 patients, while active pulmonary tuberculosis was present when the carcinoma was discovered in 14 patients. Activity is considered real when two or more positive cultures for acid-fast bacilli are established.

Three additional patients with bronchogenic carcinoma were eliminated from this series. There was 1 patient with a single positive culture and 16 negative cultures. The second patient had three positive smears, but no positive cultures. The third patient was dropped, even though areas of active tuberculosis were found at postmortem, because smears and cultures of his sputa were not obtained.

Age.—Ages of these 24 patients ranged from 31 years to 68 years with a mean age of 58 years. Those with active tuberculosis had a mean age of 54 years, as compared to a mean age of 62 for those classified as having inactive disease.

Sex and Race.—Of the 24 patients, 22 were white males and 2 were Negro males. These figures do not represent actual age and race incidence, because the hospital population is overwhelmingly male.

Site of the Bronchogenic Carcinoma.—Bronchogenic carcinoma and tuberculosis occurred in the same lung in 19 patients. In 16 cases (66 per cent), the two diseases occurred in the same lobe. The carcinoma developed in a site other

From the Thoracic Surgery Section, Surgical Service, Veterans Administration Medical Teaching Group Hospital, Memphis 15, Tenn.
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than that involved by tuberculosis in 8 patients. In 5 of these patients the disease had been considered inactive from 2 to 16 years, and active in 3 patients.

Duration of Tuberculosis.—Six patients with active pulmonary tuberculosis were found to have coexisting bronchogenic carcinoma within 6 months after the establishment of the diagnosis of tuberculosis. Another 6 patients were found to have carcinoma 1 to 10 years after the diagnosis of active tuberculosis was established. Two patients had had active disease for more than 10 years at the time that the carcinoma was discovered. All of the patients with inactive tuberculosis had been considered inactive for 2 to 10 years when the bronchogenic carcinoma was found.

Diagnostic Aids.—Bronchoscopy and cytology were utilized in 16 patients. Bronchoscopically, a positive biopsy was obtained in 4 patients (25 per cent), visual evidence of tumor without biopsy was seen in 5 patients (31 per cent), and a negative examination was present in 7 patients (44 per cent). Suspicious cytology was present in 2 patients (12 per cent), positive cytology in 5 patients (31 per cent), and negative cytology in 9 patients (56 per cent).

Diagnosis.—The diagnosis of bronchogenic carcinoma presents many difficulties in patients with pulmonary tuberculosis.[5] The infection usually dominates the clinical picture so that neoplasm is frequently overlooked for varying periods of time. When active tuberculosis was present, the earliest diagnosis of coexistent bronchogenic carcinoma was made in 4 patients after 3 months of hospitalization and antituberculosis chemotherapy; in 1 patient after 6 months; in 7 patients after 1 or more years of hospitalization and treatment. The diagnosis was more rapidly made in the group with inactive disease.

A diagnosis of carcinoma was never made during life in 3 patients. These 3 patients were hospitalized 4 months, 8 months, and 12 months, respectively. All patients had x-ray studies at regular intervals (at least once a month). Two patients had active tuberculosis and in a third patient the disease was considered to be inactive.

Treatment.—Seven patients were subjected to exploratory thoracotomy. In 6 of these, the neoplasms were resected, three by pneumonectomy and three by lobectomy. Two of the pneumonectomy patients and one of the lobectomy patients received ancillary treatment in the form of radiation therapy or nitrogen mustard, and 1 patient received both nitrogen mustard and radiation therapy.

Results.—All patients except 1 are now dead. The survivor had a right upper lobectomy for alveolar cell carcinoma on Sept. 24, 1956. Only 2 other patients lived longer than 10 months after the diagnosis of carcinoma was established. Both of these patients died 15 months after treatment.

DISCUSSION

From postmortem examinations of tuberculosis patients, Gerstl and his associates[6] found bronchogenic carcinoma in 0.44 per cent, while Robbins and

Silverman[7] reported an incidence of 1.5 per cent. Bronchogenic carcinoma has been found in 0.38 per cent of the patients with active tuberculosis seen during the past 8 years at the Mississippi State Sanatorium.[8] The series reported here represents an incidence of 0.2 per cent. This is 20 times greater than the incidence reported in the general population and is comparable to that reported for heavy smokers as described by Pack and Ariel.[9] The autopsy incidence reported in 1946 by Gerstl[6] was 0.44 per cent, while the incidence reported for the general population was only 0.0058 per cent. These figures denote a higher bronchogenic carcinoma rate in patients with pulmonary tuberculosis. Inasmuch as tuberculosis presently tends to appear more frequently in the older patients (over the age 45), these two diseases can be expected to occur together more frequently in the future.[10, 11]

Since the therapeutic results in this series were extremely poor, a search of the clinical records and x-ray films was made to ascertain if there were any clinical or radiologic evidences which would have permitted earlier recognition of the malignancy.

Some of the following clinical and radiologic findings should make one suspicious of a possible coexistent neoplasm in tuberculosis patients: (1) if, after a reasonable trial of antituberculosis chemotherapy, there is no improvement of the patient's general and radiologic status, (2) enlargement of the hilar area, especially if unilateral,[12, 13] (3) atelectasis of a lobe or segment of the lung,[13, 14] (4) solitary nodular density, especially if it is in the lower lobes and has a fuzzy border, and (5) cavities with a thick, irregular wall and a poorly visualized, eccentrically placed, radiolucent central area.

SUMMARY

1. A study of case histories of 11,000 patients with tuberculosis and 650 patients with bronchogenic carcinoma was made in an attempt to establish the relative incidence of the coexistence of these two disease processes in the same patient.

2. The findings indicate that this combination of diseases occurs 20 times more frequently in the patient with tuberculosis than in the patient from the general population.

3. The study suggests that this incidence may increase as the age of the tuberculous patient becomes older.

4. All male patients with tuberculosis over the age of 45 should be studied frequently by clinical and radiologic examinations in the quest for location of the malignant lesion, in addition to the infection. Any suspicion should then invoke a complete investigation to prove the existence or nonexistence of the two lesions.

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BILATERAL SIMULTANEOUS SQUAMOUS CELL CARCINOMA OF THE LUNG

Clarence I. Britt, M.D., A. J. Christoforidis, M.D., and Neil C. Andrews, M.D., Columbus, Ohio

CARCINOMA of the lung is one of the most common tumors of the adult male. The occurrence of bilateral simultaneous primary squamous cell carcinoma arising from major bronchi in a living patient is unknown.[1] Individual cases have been reported in which pulmonary resection was carried out for carcinoma of the lung and years later a malignancy was found in the other lung.[2] Cases have been reported in which carcinoma in situ, a suspected precursor of cancer of the lung, has been found in areas of the same or the opposite lung after pulmonary resection for frankly invasive carcinoma.[3, 4] The presence of squamous cell carcinoma in one lung and a small cell or oat cell carcinoma in the opposite lung has been reported.[1] In all of these cases the diagnosis of bilateral pulmonary malignancy was made partially, if not completely, on the basis of pathologic examination of the resected specimen and examination of autopsy material. A diagnosis of bilateral simultaneous primary squamous cell carcinoma of the lung in a living patient has not been reported in the literature.

CASE REPORT

A 60-year-old white male laborer (I. C. H.) developed a cough productive of yellow-white sputum 3 years prior to admission to the hospital. Fifteen months before hospitalization he developed a cold, pleuritic pain, fever, chills, night sweats and had an increase in cough and sputum. These symptoms subsided in 2 weeks but anorexia persisted. Six months before hospitalization the patient first noted the onset of exertional dyspnea and the recurrence of cough with yellow-white sputum. Three months later he developed intermittent fever, increasing anorexia, constipation, weakness, and had a 10-pound weight loss. These symptoms progressed and 1 month before admission x-ray studies of the chest demonstrated bilateral apical "exudative" pulmonary disease. Sputum examinations were negative for acid-fast bacilli but the roentgenogram was so suggestive of pulmonary tuberculosis that he was admitted to the Ohio Tuberculosis Hospital for diagnosis and treatment.

A roentgenogram of the chest made 7 years prior to admission was reported as normal. The patient smoked one package of cigarettes per day, was treated for syphilis in 1952, and had a 40-year history of alcoholism requiring hospitalization on three occasions. He denied alcoholic intake for the previous 3 years.

From the Ohio Tuberculosis Hospital; the Division of Thoracic Surgery, Department of Surgery; and the Department of Radiology, the Ohio State University Health Center, Columbus, Ohio.

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The patient was a well-developed white man with normal vital signs, who appeared chronically ill. There were normal breath sounds over both lung fields and no râles were heard. The liver was firm, nodular, nontender, and enlarged 2 fingerbreadths below the right costal margin. The prostate was enlarged, soft, and nontender on rectal examination. The remainder of the physical examination was within normal limits.

Admission hemoglobin was 11.2 Gm. per cent, hematocrit 39 per cent, white blood cell count 6,850 cells per cubic millimeter with a normal differential. Urinalysis and blood chemistries were normal. Histoplasmosis skin test was positive 20 by 20 mm. PPD No. 1, intermediate, PPD No. 2 and old tuberculin skin tests for tuberculosis were negative. Serologic test was positive for syphilis. Sputum smears and cultures for tuberculosis were negative.

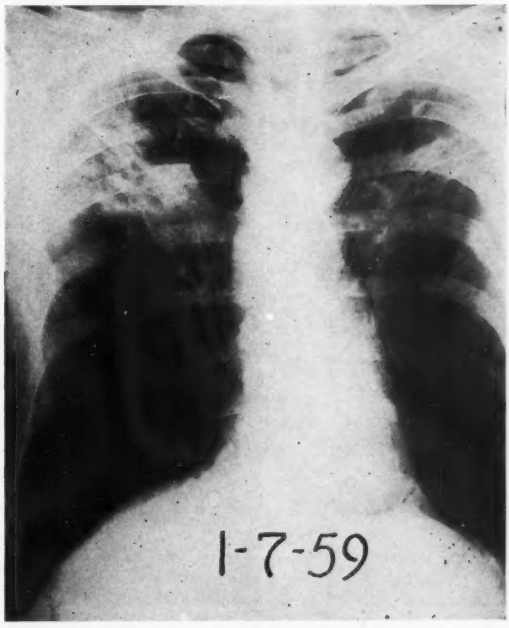


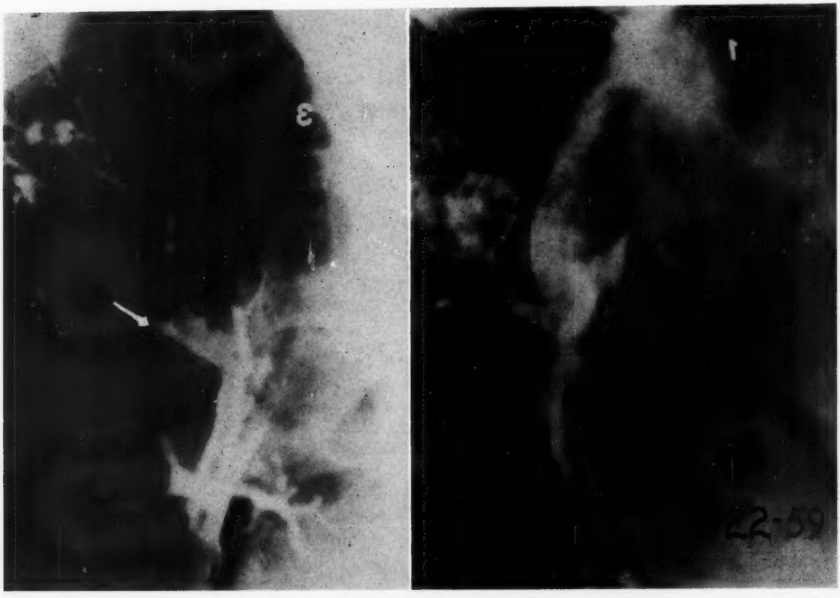
Fig. 1.—Note the bilateral hilar lymph node enlargement, the soft tissue mass above the right hilum, and linear atelectatic changes in both infraclavicular areas.

The pre-admission chest roentgenogram on Nov. 20, 1958, demonstrated a soft tissue mass in the right infraclavicular area adjacent to the hilum. Linear and nodular infiltrations were present in both upper lobes. Both hila showed lymph node enlargement. Additional findings were aortic arch calcification, healed fractures of the left sixth and seventh ribs, and left pleurodiaphragmatic adhesions. A roentgenogram of the chest on Jan. 7, 1959, showed the previously described infiltrations in the infraclavicular regions of both upper lung fields (Fig. 1). The soft tissue mass adjacent to the right hilar area was again noted, with bilateral lymph node enlargement, but with no evidence of cavitation.

A bronchoscopy on Jan. 14, 1959, showed the left lower lobe bronchus and its segmental bronchi to be normal. The left upper lobe bronchus appeared normal for 1 cm. when visualized through a right angle telescope. The right lower lobe and middle lobe bronchi were also normal. Thick green purulent secretions were seen obstructing the

anterior segmental bronchus of the right upper lobe bronchus. Aspiration of this material did not result in complete clearing of the area of obstruction. A biopsy from the right upper lobe carina was reported to be normal bronchial tissue with squamous metaplasia. Biopsy of the left upper lobe carina showed slight chronic nonspecific bronchitis. Culture of the aspirate was negative for acid-fast bacilli but a moderate growth of hemolytic *Staphylococcus albus* and streptococci was obtained.

A bilateral bronchogram on Jan. 22, 1959, demonstrated obstruction of the posterior and anterior segments of the right upper lobe with partial atelectasis corresponding to the obstructed segments (Fig. 2, *A*). The apical segment of the right upper lobe and the segmental bronchi of the right middle and lower lobes were well outlined. On the left side there was complete obstruction of the left upper lobe bronchus, including the lingula (Fig. 2, *B*). All bronchi of the left lower lobe were well visualized. Other changes in both lung



A. *B.*

Fig. 2.—*A*, Bronchogram of the right side shows obstruction of the posterior and anterior segments of the right upper lobe. Only the apical segment is patent. *B*, Note the complete obstruction of the left upper lobe bronchus (*arrow*).

fields were compatible with chronic bronchitis and emphysema. A repeat bronchogram was performed following bronchoscopic aspiration of mucus plugs and aerosol therapy to rule out the possibility of tumor. The same areas of bronchial obstruction were demonstrated which indicated probable bilateral bronchogenic tumors.

On Feb. 2, 1959, a second bronchoscopy was performed to obtain bronchial secretions for cytologic examination. The right upper lobe segmental bronchi were successfully aspirated and an intrabronchial lesion could not be demonstrated. The left main bronchus was narrowed and edematous. The left upper lobe bronchus was visualized and the anterior segmental bronchus aspirated, revisualized, and an obstruction was not seen. Generous biopsies were obtained from the right upper lobe carina and the left upper lobe carina. The bronchial secretions were collected separately during the procedure from the right main bronchus, then from the left main bronchus, and examined for the presence of malignant cells.

Microscopic examination of the biopsy specimen from the right upper lobe carina demonstrated squamous cell carcinoma in the bronchial submucosa. The biopsy from the

left upper lobe bronchus was unsatisfactory and consisted of cartilage. The bronchial aspirate demonstrated anaplastic tumor cells in the bronchial secretions from both sides.

On Feb. 11, 1959, bronchoscopy was carried out again to obtain a biopsy from the left upper lobe carina. There was no evidence of tumor visualized on the right side and no significant changes were noted. The segmental bronchi of the left upper lobe bronchus were again partially occluded with green exudate in the anterior segmental bronchus. This was aspirated and an obstruction was not visualized. A generous biopsy of the left upper lobe carina was obtained from multiple sites and submitted for microscopic examination. This specimen demonstrated poorly differentiated squamous cell carcinoma in the submucosa of the left upper lobe carina.

The diagnosis of bilateral simultaneous primary squamous cell carcinoma of the lung was made and the patient was treated with Chloromycetin for 2 weeks to improve the pneumonitis distal to the bronchial obstruction. A course of x-ray therapy was advised on an outpatient basis for palliative treatment. The possibility of pulmonary resection was discussed at some length but was thought inadvisable in view of the finding of tumor in the submucosa of both upper lobe carinas. It was felt that curative resection would not be feasible in this patient.

DISCUSSION

Bilateral simultaneous primary invasive squamous cell carcinoma of the lung diagnosed in a living patient is most unusual and has not been reported. Howard and Williams[1] recently described a patient with squamous cell carcinoma of the right main bronchus treated by pneumonectomy who had carcinoma in situ in the middle lobe and right lower lobe bronchus discovered by microscopic examination of the surgical specimen. This patient died on the twenty-fourth postoperative day from a bronchopleural fistula and empyema. Examination of the autopsy material demonstrated a small microscopic area of invasive squamous cell carcinoma at the junction of the left upper lobe and the left lower lobe bronchus. Other authors[2] have reported similar cases of squamous cell carcinoma of the lung, treated by pulmonary resection, in which a squamous cell carcinoma of the lung was detected years later in the opposite lung by follow-up x-ray studies or by examination of the autopsy material. The presence of carcinoma in situ in one lung when the other has been the site of invasive squamous cell carcinoma has been reported.[3, 4]

This case report is unique in that bilateral pulmonary neoplasms were encountered which simulated a chronic inflammatory process on initial examination. A bronchogram demonstrated abrupt segmental bronchial obstructions in both upper lobes suggesting a tumor as the most likely cause of this endobronchial abnormality.[5] Bronchoscopy and bronchial biopsy demonstrated squamous cell carcinoma in both the right and left upper lobe bronchi. The position of these tumors did not allow curative resection and treatment consisted of antibiotics to decrease pneumonitis. Palliative x-ray therapy was advised if further symptoms developed.

The possibility that one of these lesions could be a metastasis from the other side was considered but thought to be unlikely for several reasons: 1. Squamous cell carcinoma of the lung rarely, if ever, develops a solitary metastasis in the contralateral lung. 2. An abrupt endobronchial obstruction usually indicates the presence of a primary bronchogenic carcinoma.[5] 3. If one lesion represented metastasis by lymphatic extension, a positive bronchial biopsy would have been

expected to show involvement of the submucosal lymphatics on the first examination. However, normal bronchial mucosa was obtained from both the right and left upper lobe bronchi at the time of the initial biopsy, indicating that submucosal spread of the tumor had not occurred across the carina to the opposite side.

The presence of bilateral, central pulmonary lesions with abrupt endobronchial obstruction and normal intervening bronchial mucosa certainly suggests the presence of bilateral, simultaneously occurring, primary squamous cell carcinoma of the lung.

SUMMARY

1. The case history of a patient with bilateral, simultaneous primary squamous cell carcinoma of the lung is presented.

2. Bronchography demonstrated bilateral bronchial obstruction suggesting malignancy rather than infection.

3. The diagnosis was substantiated by bronchoscopy and bronchial biopsies which showed the presence of bilateral invasive carcinoma of the lung in a living patient.

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AN UNCLASSIFIABLE TUMOR OF THE ESOPHAGUS

A Case Report

Victor Hay-Roe, M.D., Rogers Lee Hill, M.D., and
W. Harold Civin, M.D., Honolulu, Hawaii

LANE[1] recently reported a series of cases of peculiar stromal change associated with squamous cell carcinoma. He reviewed no esophageal cases, but Stout and Lattes[2] discussed and illustrated 2 similar cases in the Armed Forces Institute of Pathology Fascicle on Tumors of the Esophagus. At about this time we were puzzling over an unusual esophageal lesion.

CASE REPORT

J. L. K., a 56-year-old Hawaiian man, was first seen in January, 1957, complaining of pain in the right side of his throat, progressive dysphagia, hematemesis, and marked loss of weight.

Past history revealed treatment for pulmonary tuberculosis from 1950 to 1955. He had also been treated for syphilis in the past.

Physical examination revealed a poorly-nourished man who showed evidence of recent weight loss. X-ray studies of the chest showed considerable interstitial fibrosis and extensive pleural thickening on the right side. Esophagoscopy disclosed a large necrotic mass of what appeared to be impacted food, 20 cm. from the incisors. Biopsies were reported as showing "sarcoma, type undetermined."

The patient was prepared for esophagectomy and, through an upper abdominal incision, the stomach and lower esophagus were mobilized. A right thoracotomy disclosed a lung densely adherent to the chest wall, and much bleeding was encountered in mobilizing it. The esophagus was freed and the tumor palpated within it, just inferior to the aortic arch. During the procedure, the blood continued to ooze profusely from the right thoracic cage and the patient required multiple transfusions. His blood pressure dropped progressively and became unobtainable in spite of ten pints of blood, several of which were pumped in under pressure, and it was thought that the surgery should be terminated as rapidly as possible. Since the tumor could be moved up and down within the esophagus and was attached by only a small pedicle, a longitudinal esophagotomy was made and the tumor extruded from the esophagus. The pedicle was less than 1 cm. in diameter and was attached just below the arch of the aorta. A segment of esophageal wall, 2 cm. in diameter, surrounding the base of the pedicle was excised and the esophagotomy and the chest were closed, with two chest drainage tubes inserted through separate stab wounds. These were connected to water-seal suction. No evidence of metastases was found during the procedure. At the termination of the surgery the blood pressure ranged from 40 to 60 mm. Hg systolic but, with transfusion of 1,500 c.c. of blood, it gradually returned to normal levels. The patient made an uneventful recovery.

From the Departments of Surgery and Pathology, The Queen's Hospital, Honolulu, Hawaii.
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Pathologic Report (1).—The specimen consisted of an irregularly lobulated, ovoid, gray mass of soft tissue weighing 88 grams and measuring 10 cm. in greatest dimension. Microscopic examination revealed what was interpreted as a grade 2 leiomyosarcoma and a grade 3 squamous cell carcinoma (so-called collision tumor). This lesion was later reviewed

Fig. 1.

Fig. 2.

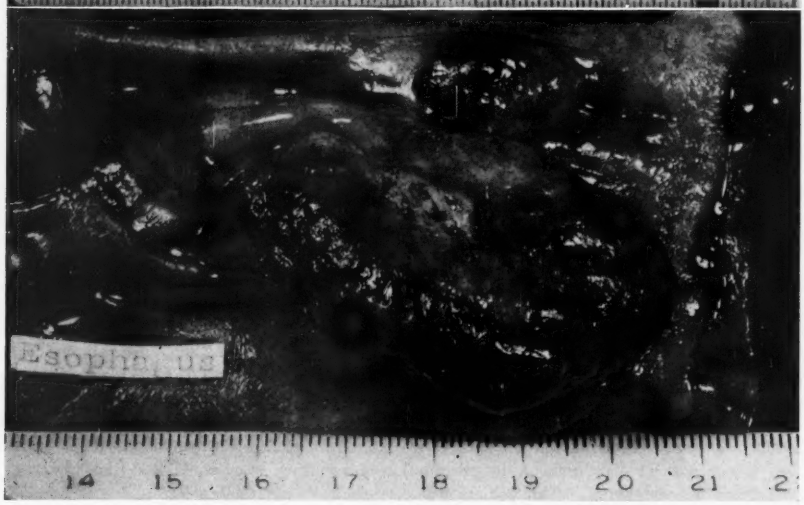

Fig. 1.—Resected segment of esophagus with sessile mass in the center and polypoid mass at one edge of the resection.

Fig. 2.—Close-up of polypoid mass near one edge of resection. The smaller polypoid mass extending from the base of the larger one can be seen above the large one in the photograph. At the left hand side of the photograph is a portion of the sessile mass.

and the possibilities of a leiomyoma with an associated squamous cell carcinoma, a carcinoma with pseudosarcomatous stromal alteration, or a carcinoma with areas resembling a sarcoma were also entertained.

The patient was ambulatory and able to eat soft foods on the fourteenth postoperative day, and he was discharged.

Because of the inadequacy of the surgery performed, the possibility of further surgery was suggested, but the patient refused. He gained weight and did fairly well for several months, but in January, 1958, he returned complaining of mild dysphagia and recent weight loss. Esophagograms at this time revealed two filling defects, one in the upper esophagus,

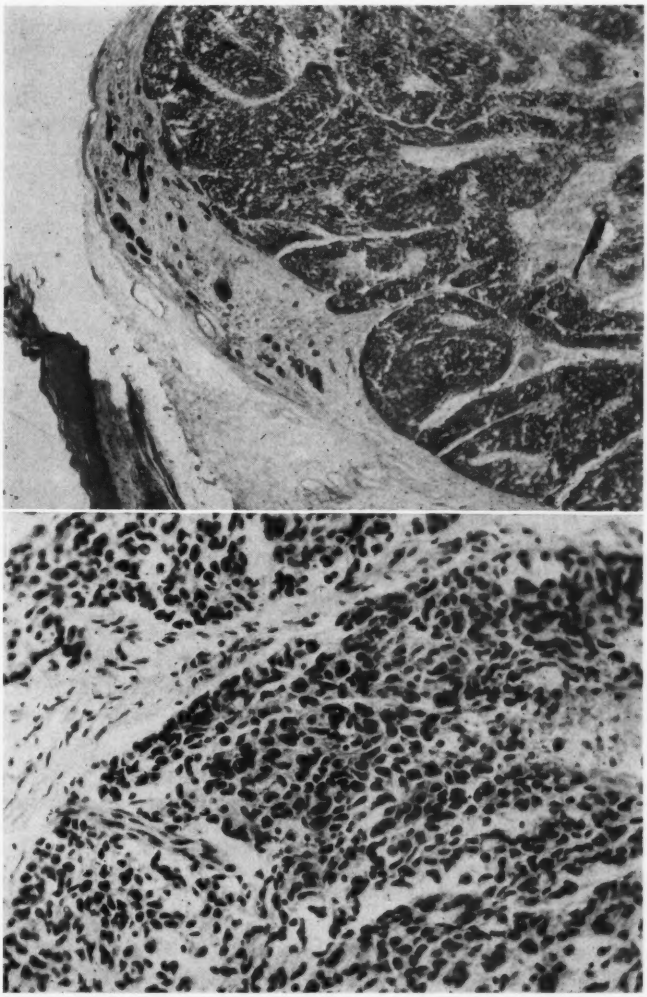

Fig. 3.—Photomicrograph showing masses of grade 3 squamous cell carcinoma invading esophageal wall. (Hematoxylin and eosin. *A*, ×45–*B*, ×265; both reduced ⅙.)

the other just above the cardia. Esophagoscopy and biopsy proved the presence of recurrent tumor, and the patient then consented to further surgery (exactly 1 year following the first operation).

He was readmitted on Feb. 18, 1958, and a radical subtotal esophagectomy was carried out through the left chest, using a combined cervical, thoracic, and abdominal incision. The stomach and duodenum were mobilized and the gastric cardia was anastomosed to the remaining esophageal segment in the neck. Careful examination revealed one area in which

the wall of the esophagus was indurated, but no spread through the wall could be detected. Critical exploration of the abdomen (including liver biopsy) and thorax revealed no evidence of metastases.

Pathologic Report (2).—The specimen consisted of a segment of esophagus measuring 21 cm. in length and weighing 105 grams (Fig. 1). Five centimeters from one end was a polypoid growth, 5 cm. in diameter (Fig. 2), which was attached by a narrow pedicle, approximately 1 cm. in diameter. At the base of this mass was another small pedunculated daughter growth, 1 cm. in diameter.

About 5 cm. from the other end, and extending to the mid-portion of the esophagus, was a sessile, nodular ulcerated growth which measured 1.5 to 2.5 cm. above the mucosa.

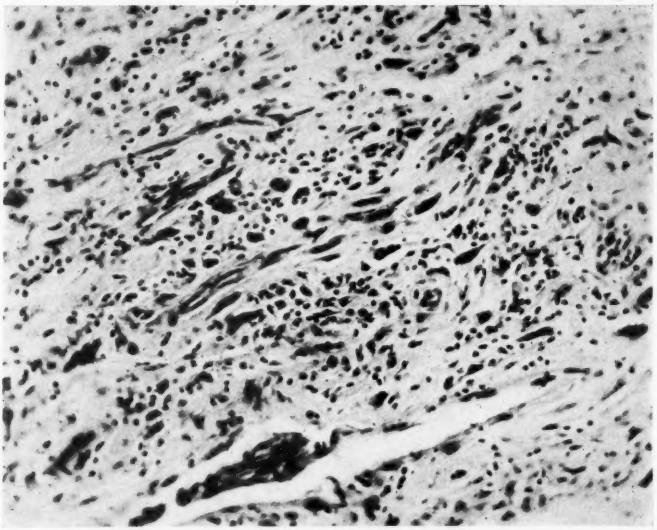

Fig. 4.—Medium-power view of anaplastic stromal giant cells. (Hematoxylin and eosin ×265.)

Microscopically, the major part of the sessile tumor was composed of nests, masses, and columns of squamous cells (Fig. 3) with variably chromatic and pleomorphic nuclei, and cells showing anisonucleosis growing in the thickened submucosa and superficial muscularis propria. Where the mucosa was not ulcerated, the covering squamous epithelium was entirely normal and there was no evidence of surface origin of the lesion which appeared to have spread submucosally. No stromal change was seen. No lymphatic channels were noted to contain tumor emboli.

The pedunculated tumor and its offshoot were composed of a core of round and spindle-shaped cells with hyperchromatic oval nuclei. Cytoplasm was variable in amount, and cell membranes were often distinct, although at times indistinct. A large number of multinucleated giant cells (Fig. 4), not of the foreign body type, was present also. There was some infiltration by lymphocytes and plasma cells. The surface of the tumor was represented by a squamous cell carcinoma showing some superficial invasion and ulceration.

It is interesting to note that although known malignancy had been present for over a year at the time of the second operation, no metastases could be demonstrated. Even infiltration of the esophageal wall was not extensive. The lower mass may have been present, although certainly less extensive, at the first operation. However, it may not have been present then.

Following surgery the patient did poorly; he was dyspneic and slightly cyanotic with marked decrease in pulmonary capacity. He became rapidly worse and died on the first postoperative day. Death was probably a result of markedly reduced pulmonary capacity incident to an extensive fibrosis from an old tuberculosis.

DISCUSSION

This case illustrates an unusual esophageal tumor of a type which has been infrequently described in the literature. Lane,[1] in Stout's laboratory, reported several cases of similar tumor occurring in the mouth, pharynx, and larynx. These polypoid growths he designated as pseudosarcomas occurring in association with a squamous cell carcinoma. In his opinion, factors present in the connective tissue adjacent to a squamous cell carcinoma could stimulate the proliferation of connective tissue, resulting in a large polypoid growth with an inconspicuous squamous carcinoma at its base. He likens the proliferation of connective tissue to that seen following trauma or irradiation, as in the "granulation tissue sarcoma" mentioned by Ewing.[3]

There seemed to be four possibilities concerning the lesion in this case. It might have been (1) a carcinoma with pseudosarcomatous change in the stroma; (2) a malignancy of the collision type; (3) a carcinoma and a benign stromal tumor, as Pearlman[8] thought some so-called carcinosarcomas were, or (4) an anaplastic carcinoma. A check for myofibrils revealed none and many nuclei appeared adjacent to the extranuclear material and not surrounded by this, so we veered away from a diagnosis of leiomyoma.

The clinical course and apparent lack of metastases seemed against the diagnosis of anaplastic carcinoma. Also, because of lack of mitoses of the stromal elements and the rarity of occurrence, a malignant stromal tumor appeared unlikely. In favor of pseudosarcomatous change was the absence of deep invasion by the stromal element, although the squamous cell carcinoma extended into the muscle wall. Pseudosarcoma was also suggested by the ability of the tumor to propagate in tissue culture on numerous transplants as polygonal and spindle elements, depending on where the tissue was taken. These former cells also showed spindling of the edges of the transplants (Fig. 5). These constituents were maintained by reculture every week or so for a period of 5 months.

The emphasis placed on tissue culture in the diagnosis depends, of course, on one's teaching and experience. Autonomous growth, which this tumor displayed, is considered as a sign of malignancy by many; thus the stromal element might be considered as malignant. There is a marked tendency, however, for epithelial elements to spindle in tissue culture and this could be the explanation of the spindling.

Stout and Lattes[2] have illustrated 2 similar cases but made no conjecture as to the mechanism of development. The authors stressed confusing the lesion with sarcoma and they also stressed that an intramucosal and occasionally an invasive squamous cell carcinoma is always present.

A number of lesions called carcinosarcomas of the esophagus of polypoid character in the literature showed no metastases of stromal elements.[4-15] The

possibility that some of these were carcinomas with pseudosarcoma has been entertained by both Stout and Lattes[2] and Lane.[1] Metastases occurred chiefly in polypoid lesions incompletely treated or of long duration, a point to be considered in the treatment of such lesions.

The question as to whether or not one can differentiate lesions in which the connective tissue element will metastasize from those in which it will not has not been resolved. Most such decisions would seem to be purely academic, because polypoid lesions with epithelial malignancy and stromal atypism, regardless of their origin, should be treated early and extensively since they often do not carry the dismal connotation of the usual squamous cell carcinomas of the esophagus. The usual squamous cell carcinoma ulcerates and infiltrates slowly although it gains access to lymphatics and metastasizes early. It does

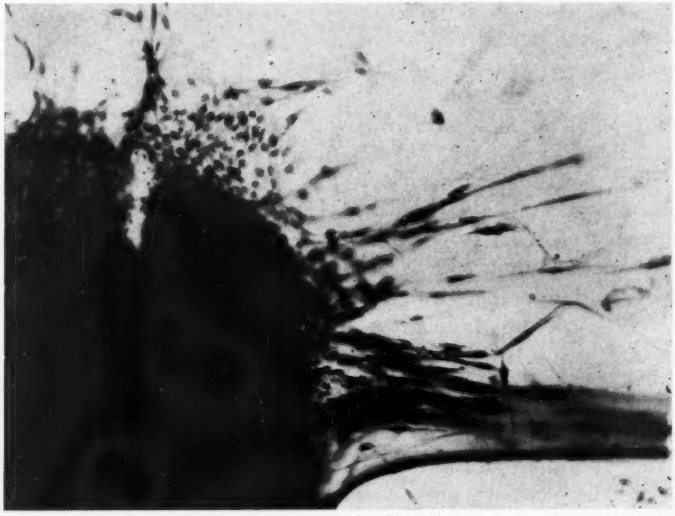

Fig. 5.—Photomicrograph of tissue culture growth in transplant which shows tendency of epithelial cells to spindle at the edges of the growth. (Hematoxylin and eosin ×120; reduced 1/6.)

not give rise to symptoms until extensive infiltration of the esophageal wall (and often the adjacent mediastinal structures and lymph nodes) brings on dysphagia and weight loss. In contrast, the rarer group of malignancies represented by this case, by nature of their exophytic growth, usually cause dysphagia early and bring the patient to the physician while the lesion is still amenable to surgery. Furthermore, the intrinsic malignancy of the polypoid lesion appears less. Bad results seem to be related to inadequate treatment.

If, on biopsy of a polypoid tumor of the upper respiratory or digestive systems, a peculiar sarcoma-like (sometimes called sarcoma) proliferation of the stroma is noted, such a finding should alert the surgeon and the pathologist to the possible presence of an adjacent squamous cell carcinoma. Since the death of this patient the authors have been able to prove that such was the case in a tumor of the nasopharynx. An aggressive approach may be lifesaving.

SUMMARY

An unusual tumor of the esophagus, showing both epithelial and connective tissue elements and differing from the usual esophageal malignancy, is reported.

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AN UNUSUAL VARIATION OF CONGENITAL TRACHEOESOPHAGEAL FISTULA

Case Report

Ira S. Goldenberg, M.D., New Haven, Conn.

CONGENITAL anomalies can frequently be explained on the basis of known embryological development. Some of the more complex structural defects such as tracheoesophageal fistula, however, are not understood completely so that conjecture and surmise play important roles in ascertaining development. Despite lack of complete knowledge, a classification of known types of tracheoesophageal fistula has been accepted generally (Fig. 1). Variants from this grouping are uncommon. Such variants may sometimes corroborate previous ideas regarding development or they may confuse the situation further. The present report describes an interesting variation of tracheoesophageal fistula.

CASE REPORT

A male infant was delivered at another hospital on June 22, 1959, after a full-term, uneventful pregnancy. Labor and delivery were unremarkable. The mother was Rh negative, the father Rh positive, and the infant Rh negative. Two siblings were without congenital abnormalities.

During the first day of life, constant suctioning of the oropharynx was necessary because large amounts of mucus were produced which interfered with the airway. When the child was 12 hours old the diagnosis of esophageal atresia with probable tracheoesophageal fistula was made after appropriate radiological studies. Thoracic hemivertebrae were also demonstrated on these films. The child was transferred to this hospital where exploration was carried out through a right thoracotomy. Continuity of a dilated esophagus was noted. Dissection of the trachea from the esophagus revealed a fistula to be present between trachea and esophagus high in the chest at the thoracic inlet. This was divided in routine fashion and the trachea closed with some difficulty because of the location in the low neck. A tube passed from the nasopharynx could be palpated in the esophagus distal to the esophageal opening but could not be passed through the opening. Esophagostomy at a lower level was then performed and a second catheter passed from below. This catheter emerged through the previous fistulous connection with the trachea. It was then apparent that a septum was present which divided the esophagus with a fistulous connection to the trachea inferior to it. The esophagus was then opened in its mid-portion and the septum divided. The esophagostomy openings were closed.

The postoperative course was benign during the first few days after operation. Oral nutrition was started on the fifth postoperative day and went well for 24 hours. An episode of aspiration of a feeding occurred on the second day of feeding and severe pneumonitis

From the Department of Surgery, Yale University School of Medicine, New Haven, Conn.
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developed. Feedings were continued, however, and were uneventful until the eighth postoperative day when another aspiration episode took place. Radiographic examination of the esophagus, using thin barium, was done and demonstrated patency of the esophagus with free flow to the stomach. Aspiration of barium occurred during the examination which outlined the trachea, demonstrating a possible fistula to the esophagus at the level of the first thoracic vertebra. A gastrostomy was then done for feeding purposes. The child took the nutrition per gastrostomy well for 9 days when another episode of aspiration occurred. Following this, the pneumonia became more marked and several episodes of cyanosis were noted.

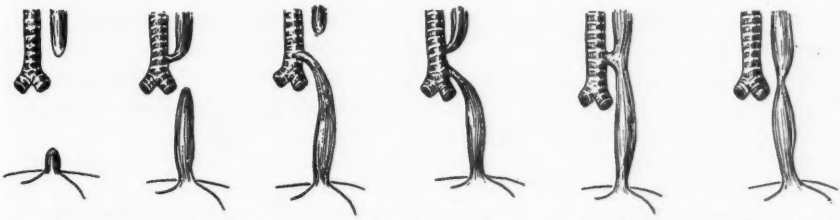

Fig. 1.—The common types of congenital abnormalities of the esophagus (from Gross[1]).

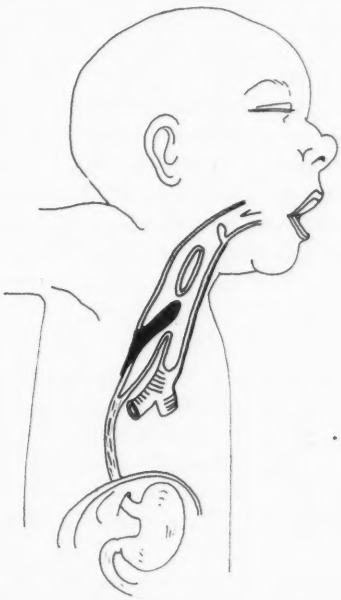

Fig. 2.—A diagrammatic representation of the patient reported here. The esophagus and trachea have been cut open to show the esophageal septum (solid black) and its relation to the fistulas.

The general condition of the child deteriorated. On the twenty-third day of life a cardiac murmur was heard for the first time and it was evident that congestive cardiac failure was present. Digitalis was given but rapid worsening in the child's condition followed and he died on the twenty-eighth postoperative day.

Postmortem examination revealed the area of repair to be intact without evidence of leak. Meticulous examination of the specimen revealed a second, minute tracheoesophageal fistula in the neck, about 1.5 cm. above the repaired one. This second fistula was so small as to have been overlooked during the preliminary appraisal of the organs (Fig. 2).

Marked hypertrophy of the right heart was present with interauricular and interventricular septal defects and a bicuspid aortic valve. Coarctation of the aorta and a patent ductus arteriosus were present, as was marked hydrocephalus.

Death was attributed to right heart failure. Acute cor pulmonale resulting from the marked pneumonia and consolidation of the lungs probably caused decompensation of the congenitally abnormal heart.

DISCUSSION

Unusual features in the patient presented here are (1) the septum in the esophagus associated with (2) the high location of the fistula to the trachea which was closed at operation and (3) the presence of a second tracheoesophageal fistula in the neck which was noted at autopsy. This infant resembled a child reported by Singleton and Knight[2] in whom two fistulas were present—the first repaired successfully, the second found in the neck at postmortem examination.

A review of the normal development of the trachea and esophagus offers some suggestions as to the development here. During the fourth week of gestation, the primordial respiratory tract anlage appears from the ventral side of the foregut. This outgrowth separates from the gut by the growth of bilateral longitudinal ridges which fuse. The fusion extends cranialward while the tracheal portion grows caudally rapidly. Some time before the seventh week of development it is believed that the esophageal lumen is obliterated by epithelial proliferation to recanalize during the eighth week. It is possible that the septum in the patient here represented a persistence of the obliteration of a segment of the esophageal lumen. Variation in growth rate of esophagus and trachea could account for the fistulas although it is difficult to explain the higher than usual location of them.

SUMMARY

A variation of congenital tracheoesophageal fistula has been presented in which there was esophageal continuity, a mid-esophageal septum, and two separate fistulas to the trachea. Autopsy following death on the twenty-eighth day of life disclosed additional abnormalities: hypertrophy of the right heart, interauricular and interventricular septal defects, bicuspid aortic valve, coarctation of the aorta, patent ductus, and marked hydrocephalus.

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BRONCHOGRAPHY THROUGH PUNCTURE OF THE CRICOTHYROID MEMBRANE

Gerald B. Reams, Captain, USAF (MC), and Morton A. Bosniak, Captain, USAF (MC),** Wimpole Park, England*

IN VIEW of the (1) inconsistent radiologic results, (2) toxic anesthetic reactions, (3) technical difficulties, and (4) discomfort to the patient following any of the laryngeal catheterization methods by the oral or nasal route, it was decided to evaluate the method of bronchography, as originally suggested by Sicard and Forestier in 1922, which has been used much more frequently on the continent and in England than in this country. Numerous advantages and disadvantages appeared to us in our review of the literature and in our original consideration of this technique. The advantages appeared to be that, without the irritating presence of a catheter, the amount of local anesthetic necessary would be greatly reduced since only the subglottic portion of the tracheobronchial tree would require anesthesia. This in turn should decrease the number of toxic reactions as well as increasing the patient comfort and safety. Since the cough reflex as initiated by laryngeal and carinal stimulation was eliminated, it was believed that more constant radiologic results could be obtained. Technically the necessary positioning of the patient during the procedure, in order to obtain complete segmental filling, appeared to us to be a possible inherent weakness in this technique. Other disadvantages considered were the possibility of damage to the trachea, perforation of the esophagus, extravasation of the dye into the paratracheal tissues, the possibility of subcutaneous and mediastinal emphysema and infection, air embolism, or breakage of the needle in the trachea.

HISTORICAL DATA

Bronchography was originally performed by Chevalier Jackson[1] in 1905 by means of insufflation of a bismuth subcarbonate compound through the bronchoscope. Sicard and Forestier[2] reported the use of Lipiodol by means of cricothyroid puncture in 1922. Numerous other technical methods have been used for the introduction of the bronchographic medium; the introduction of the nasal catheter into the trachea under topical anesthesia has been the most

From the 7510th USAF Hospital, Wimpole Park, England
Received for publication Nov. 10, 1959.
*Chief, Department of Surgery.
**Chief, Department of Radiology.

widely used method in the United States. Due to its slow absorption and possibility of tissue reaction, Lipiodol has now been supplanted, first, by a partial transition to the water-soluble bronchographic compounds which were not completely satisfactory, and now to Dionosil which is an aqueous or oily suspension of a salt of the basic same acid as Diodrast. When warmed it has offered a medium of low irritation, good contrast, satisfactory viscosity, and disappears from the lungs within 24 hours. The choice of the topical anesthetic has changed from the use of cocaine originally to a now wide variety of potent agents of low toxicity. Both Xylocaine and Cyclaine were used satisfactorily during this study. The bronchopulmonary segmental anatomic terminology, as suggested by Jackson and Huber[9] in 1943, was used for conformity in this study since the patients investigated represented the combined therapeutic problems of the medical, radiologic, anesthetic, and surgical teams.

INDICATIONS

The chief value of bronchography lies in the demonstration of the presence or absence of bronchiectasis. Its use, we believe, should not be restricted to those patients with the typical symptomatology of a cough productive of large amounts of sputum but should be given serious consideration in all cases of hemoptysis as an adjunct to other more commonly used methods of diagnosis to eliminate the possibility of unsuspected upper lobe involvement in the presence of a normal routine chest film. Its use as a guide in the surgical treatment of pulmonary tuberculosis is becoming a more widespread feature. Rare congenital abnormalities, as well as foreign bodies and tumors of the bronchus, may on occasion be diagnosed only by the performance of bronchography. The etiology of pulmonary cysts may also be ascertained, and other diseases, such as asthma and emphysema, may show not only slight morphologic changes but also changes in bronchophysiologic characteristics. Since we believe this to be a procedure which offers a good percentage of useful yield with low morbidity, the variety of clinical problems as mentioned above are considered indications for bronchography.

TECHNIQUE

Premedication in these cases has varied with the use of either a barbiturate 90 minutes before the procedure combined with morphine and atropine 60 minutes before the procedure as the standard, with use of the barbiturate or narcotic alone, or no premedication in certain individual cases. The former patients naturally were less apprehensive, with slightly diminished secretions and cough reflex. The personality make-up of the individual seemed to be as important as any factor in determining the difference between a very cooperative and comfortable patient and the small percentage of others who were not. A complete and thorough explanation of the procedure appears to be of the utmost importance in obtaining patient cooperation. The patient is positioned with the head slightly extended on the fluoroscopic table with the head of the

table elevated approximately 20 degrees. The area is prepared, using Phisohex and aqueous Zepharin. Local anesthesia of the skin and cricothyroid membrane is obtained by either 1 c.c. of 1 per cent Cyclaine or 1 per cent Xylocaine. Following this, a 16-gauge needle specially prepared with a short bevel, and of a length calculated to penetrate one half of the diameter of the trachea from the skin, is inserted. Flexible polyethylene tubing is then connected to the needle and approximately 2 c.c. of 1 per cent Cyclaine is injected with the patient on his back, and 1 c.c. with the patient on his right side and 1 c.c. when on his left side. (In only one instance, early in our performance of this technique, was it necessary to reinsert the needle because of the vigorous swallowing of the patient.) After this procedure the patient is allowed to rest for several minutes

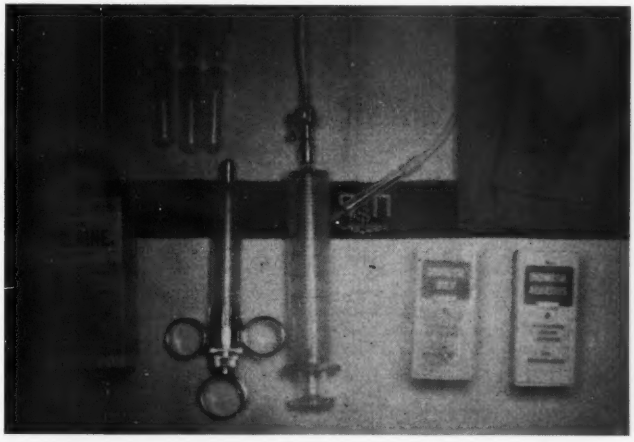


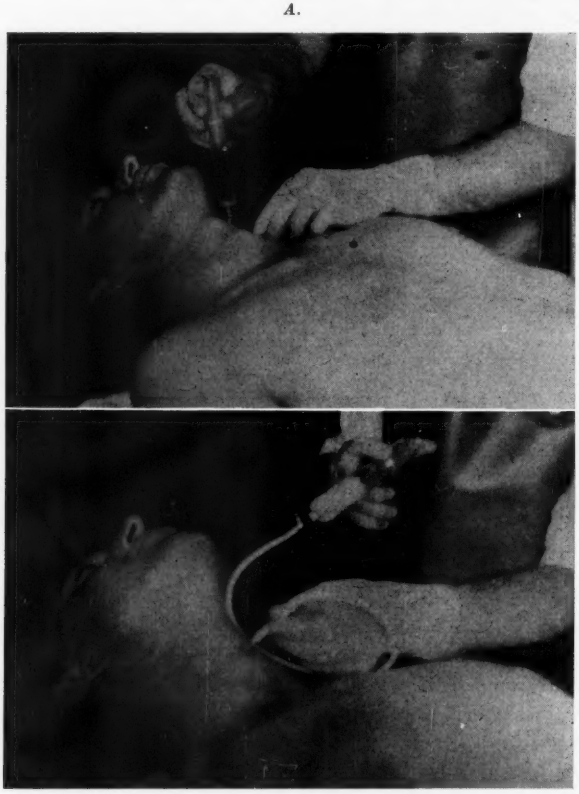
Fig. 1.—Bronchographic materials.

before, under intermittent fluoroscopic control, 15 c.c. of warmed aqueous or oily Dionosil is injected with the patient on his back and then another 5 c.c. with the patient lying on his right side, having turned approximately 120 degrees, then back on his left side through the same arc for another injection of 5 c.c. Filling is checked and, if satisfactory, films are taken at a target—film distance of 6 feet. If any segment is not filled satisfactorily he is positioned properly and another 5 c.c. is injected before completion of fluoroscopy. After these films are viewed the patient is asked to breathe deeply and cough gently several times before the final posteroanterior film is taken. We have found that on occasions this will provide somewhat of a more definite air contrast coating of the bronchi without appreciable alveolar filling. The needle is removed and a small Band-aid dressing is applied which is removed in 24 hours.

RESULTS

During the period of study, seventeen bronchograms were performed by the laryngeal catheterization method using the same type of premedication and

anesthetic agents, except that with the latter cases, even when an aersol was used, anesthesia was never completely satisfactory using less than 12 c.c. of 1 per cent Xylocaine or 1 per cent Cyclaine, and it usually required slightly more. (This is in comparison with an average of 5 c.c. when using the cricothyroid puncture technique.) In the 17 catheterization cases, there was one convulsive reaction, 1 patient with transient cyanosis, and 3 patients with mild reactions of pallor, sweating, bradycardia, and hypotension. Ninteen cricothyroid membrane puncture bronchograms were performed with the technique

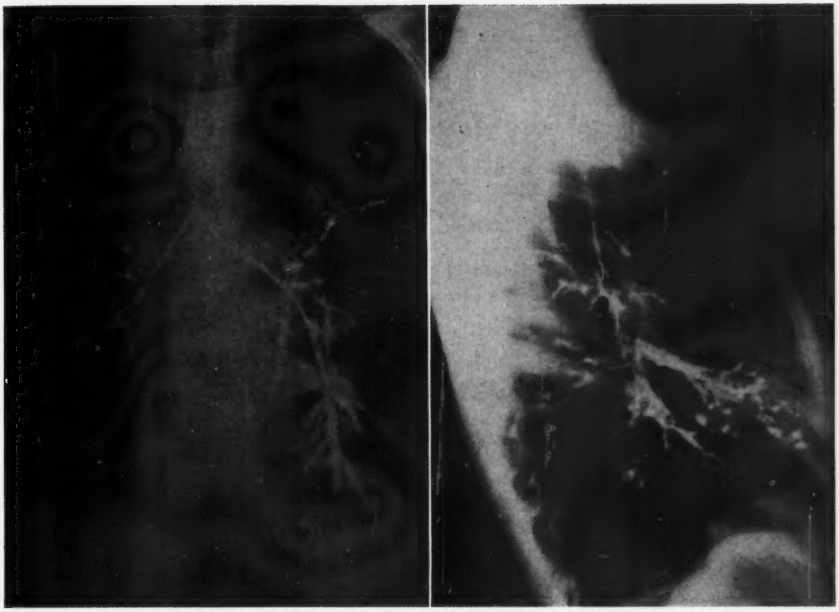


A.

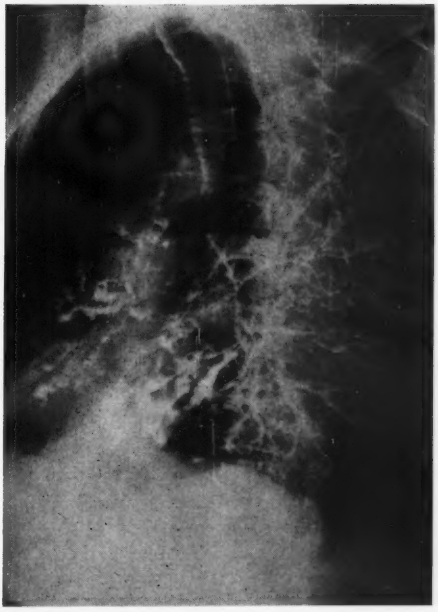
B.

Fig. 2.—Bronchographic technique. *A*, Insertion of local anesthesia. *B*, Injection of the bronchographic medium.

outlined previously with only one instance of syncope and bradycardia in this group. In an evaluation of the diagnostic quality of the films, it was considered that there was incomplete filling in 4 of the 17 cases in the first group, whereas, in the latter, there was only 1 case in which the filling was not completely satisfactory. The over-all quality was much better in the group which had had cricothyroid membrane puncture. As far as patient comfort was



A. *B.*



C.

Fig. 3.—Representative bronchograms. *A*, R.N. (No. 26501). An anteroposterior view which shows bronchiectasis of the basilar segments of the left lower lobe. *B*, A.K. (No. 24219). A lateral view which shows bronchiectasis of the middle lobe and basilar segments of the right lower lobe. *C*, M.G. (No. 25512). A right anterior oblique view which shows extensive basilar segmental bronchiectasis.

concerned, 5 patients of the second group had previously had laryngeal catheterization bronchography and all 5 expressed strong preference for the needle method. Technically, by using the method described, there was less difficulty in filling the upper lobes through the needle than had been anticipated, although the head of the table was never lowered below 10 degrees above the horizontal. There was 1 patient in whom the needle did slip out of the trachea near the end of the procedure resulting in a minimal extravasation which cleared spontaneously in 72 hours. One patient developed a nonhemolytic streptococcal cellulitis surrounding the site of the needle puncture and the same organism was cultured from the tracheobronchial tree. This responded well to appropriate antibiotic therapy and warm compresses but did prolong his hospital stay for several days beyond that which would have been required for the completion of his diagnostic workup. (Several of the films and a picture of the material necessary for the performance of this type of bronchography are included.) It is our belief that the use of a number of short beveled needles of various lengths, so that one can be chosen of the correct length to measure just that distance between the skin and the mid-diameter of the trachea, is a very important factor in the prevention of complications in this technique.

DISCUSSION

In a comparison of our limited series of patients between the nasal catheterization method with the cricothyroid puncture method, we were impressed with the advantages of the latter. In the group which had catheterization there was one convulsive reaction which responded well to an intravenous barbiturate and oxygen, and one episode of transient cyanosis. Three patients showed milder reactions of pallor, sweating, bradycardia, and hypotension, whereas, in the cricothyroid group there was only 1 case of bradycardia and hypotension which cleared spontaneously.

In the group with cricothyroid puncture, there was 1 case of minimal extravasation and 1 other in which a cellulitis developed around the injection tract which required treatment and prolongation of the hospital stay for several days.

Taking all factors into consideration, it was our belief that the latter procedure was the most satisfactory because of its decrease in anesthetic reactions and its overwhelming preference by the patients, particularly those on whom the catheterization method had been used previously. Its simplicity and ease of performance, combined with the production of much more satisfactory diagnostic films, are additional assets to be considered in the choice of methods. Our hope is that this method will be given a more widespread trial in the United States than it has had, in order to ascertain if the disadvantages mentioned earlier might not be more rare than originally anticipated.

SUMMARY

In view of the (1) inconsistent radiologic results, (2) toxic anesthetic reactions, (3) technical difficulties, and (4) discomfort to the patient following the usual catheterization method of bronchography, it was decided to evaluate

the transtracheal method, as described by Sicard and Forestier in 1922 and used more generally on the continent and in Great Britain than in the United States.

The patient is prepared for this procedure by a combination of morphine and atropine given 1 hour before the procedure with a barbiturate sometimes added, although the technique has been satisfactorily performed with no premedication. The patient is positioned on the fluoroscopic table with the neck slightly hyperextended. Local anesthesia is obtained by a total dose of 5 c.c. of the anesthetic solution (1 per cent Cyclaine or Xylocaine) and the bronchographic medium is injected through a 16 gauge needle which has been inserted through the cricothyroid membrane. It is of importance that this needle be of such length as to approximate the distance between the skin and the mid-diameter of the trachea. Dionosil is used as the bronchographic medium and bronchial filling of all segments is accomplished by positioning of the patient, being careful not to lower the head more than 10 degrees in order to prevent reflux into the unanesthetized larynx.

In evaluating this method, bronchoscopy performed by catheterization was compared with that performed transtracheally, with the latter method being the more satisfactory in our hands in alleviating the difficulties mentioned. It is hoped this technique will now be given a more widespread trial to ascertain its true place in our diagnostic armamentarium.

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The dates of the next meeting of The American Association for Thoracic Surgery were erroneously given on the cover of June Issue as May 24, 25, and 26, 1961, instead of *April* 24, 25, and 26, 1961.

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ADAMS, HERBERT D.—Lahey Clinic, 605 Commonwealth Ave., Boston 15, Mass.
ADAMS, RALPH—Huggins Hospital, Wolfeboro, N. H.
ADAMS, WILLIAM E.—University of Chicago, 950 East 59th St., Chicago 37, Ill.
ADLER, RICHARD H.—100 High St., Buffalo 3, N. Y.
ALLBRITTEN, FRANK F., JR.—University of Kansas Medical Center, Kansas City 12, Kan.
ALLEY, RALPH D.—Albany Hospital, Albany, N. Y.
ANDREWS, NEIL C.—466 West Tenth Ave., Columbus 10, Ohio
ASHBURN, FRANK S.—1835 Eye St., N.W., Washington 6, D. C.
AUERBACH, OSCAR—Veterans Adm. Hospital, East Orange, N. J.

BAFFES, THOMAS G.—Children's Memorial Hospital, Chicago 14, Ill.
BAHNSON, HENRY T.—201 Cedarcroft Road, Baltimore 12, Md.
BAILEY, CHARLES P.—New York Medical College, 1249 Fifth Ave., New York, N. Y.
BARKLEY, HOWARD T.—4414 Montrose Blvd., Houston 6, Texas
BARONOFSKY, IVAN D.—Mount Sinai Hospital, New York 29, N. Y.
BATTERSBY, JAMES S.—1040 W. Michigan St., Indianapolis 7, Ind.
BEATTIE, EDWARD J., JR.—1753 W. Congress Parkway, Chicago 12, Ill.
BEECHER, HENRY K.—Massachusetts General Hospital, Boston 14, Mass.
BELL, JOHN W.—Veterans Adm. Hospital, Seattle 8, Wash.
BENSON, CLIFFORD D.—1515 David Whitney Bldg., Detroit 26, Mich.
BERG, RALPH, JR.—231 Medical Center Bldg., Spokane 4, Wash.
BERNATZ, PHILIP E.—Mayo Clinic, Rochester, Minn.
BLACK, HARRISON—319 Longwood Ave., Boston 15, Mass.
BLADES, BRIAN—901 Twenty-third St., N.W., Washington 7, D. C.

*If any corrections should be made in the spelling of names or in the addresses of members, please notify the Administrative Assistant of the Association, Miss Ada Hanvey, 308 Carondelet Building, St. Louis (Clayton) 5, Missouri.

BLAKEMORE, WILLIAM S.---3400 Spruce St., Philadelphia 4, Pa.
BORTONE, FRANK---2765 Hudson Blvd., Jersey City 6, N. J.
BOSHER, LEWIS H.---1200 E. Broad St., Richmond 19, Va.
BOYD, DAVID P.---Lahey Clinic, 605 Commonwealth Ave., Boston 15, Mass.
BRADSHAW, HOWARD H.---Bowman Gray School of Medicine, Winston-Salem, N. C.
BRANTIGAN, OTTO C.---104 W. Madison St., Baltimore 1, Md.
BREWER, LYMAN A. III---2010 Wilshire Blvd., Los Angeles 57, Calif.
BRINDLEY, G. VALTER, JR.---Scott and White Clinic, Temple, Texas
BROWN, IVAN W., JR.---Duke University Hospital, Durham, N. C.
BROWN, ROBERT K.---1624 Gilpin St., Denver 6, Colo.
BROWNRIGG, GARRETT M.---47 Queens Road, St. Johns, Newfoundland
BRUNEAU, JACQUES---847 Rue Cherrier, Montreal 24, Canada
BUCKINGHAM, WILLIAM W.---314 Professional Bldg., Kansas City 6, Mo.
BUGDEN, WALTER F.---Medical Arts Bldg., Syracuse 10, N. Y.
BURFORD, THOMAS H.---Barnes Hospital, St. Louis 10, Mo.
BYRON, FRANCIS X.---120 S. Lasky Dr., Suite 203, Beverly Hills, Calif.

CALLAGHAN, JOHN C.---502 Medical Arts Bldg., Edmonton, Alberta, Canada
CARLSON, ROBERT I.---1410 West Michigan St., Indianapolis 2, Ind.
CARR, DUANE---20 S. Dudley St., Memphis 3, Tenn.
CARTER, MAX G.---670 George St., New Haven, Conn.
CHAMBERLAIN, JOHN MAXWELL---23 East 79th St., New York 21, N. Y.
CHAMBERS, JOHN S., JR.---2850 Sixth St., San Diego 3, Calif.
CHESNEY, JOHN G.---1550 N.W. 10th Ave., Miami 37, Fla.
CLAGETT, O. T.---Mayo Clinic, Rochester, Minn.
CLATWORTHY, H. WM., JR.---Children's Hospital, 17th St. at Livingston Park, Columbus 5, Ohio
CLOWES, GEORGE H. A., JR.---Cleveland Metropolitan General Hospital, Cleveland 9, Ohio
COHN, ROY B.---Stanford University Hospital, San Francisco 15, Calif.
COLEMAN, FRANK P.---1111 W. Franklin St., Richmond 20, Va.
CONDON, WILLIAM B.---1825 Gilpin St., Denver 18, Colo.
CONKLIN, WILLIAM S.---1006 Standard Ins. Bldg., Portland 5, Ore.
COOLEY, DENTON A.---Baylor University College of Medicine, Houston 25, Texas
COTTON, BERT H.---1321 N. Vermont Ave., Los Angeles 27, Calif.
CRANDELL, WALTER B.---Veterans Adm. Hospital, White River Junction, Vt.
CRAWFORD, E. STANLEY---1200 M. D. Anderson Blvd., Houston 25, Texas
CREECH, OSCAR, JR.---Tulane University School of Medicine, New Orleans 12, La.
CRIMM, PAUL D.---Boehne Hospital, Evansville 12, Ind.
CROSS, FREDERICK S.---11311 Shaker Blvd., Cleveland 4, Ohio
CURRERI, ANTHONY R.---1300 University Ave., Madison 6, Wis.
CUTLER, PRESTON R.---535 East 1st South, Salt Lake City 2, Utah

DAILEY, JAMES E.---4109 Montrose Blvd., Houston 6, Texas
DAMMANN, JOHN F.---632 Preston Place, Charlottesville, Va.
DANIEL, ROLLIN A.---410 Medical Arts Bldg., Nashville 12, Tenn.
DANIELS, ALBERT C.---490 Post St., San Francisco 2, Calif.
DAUGHTRY, DEWITT C.---1550 N.W. 10th Ave., Miami 37, Fla.
DAVILA, JULIO C.---269 S. 19th St., Philadelphia 3, Pa.
DAVIS, EDGAR W.---1150 Connecticut Ave., Washington 6, D. C.
DAY, J. CLAUDE---307 David Whitney Bldg., Detroit 26, Mich.
DE BAKEY, MICHAEL E.---Baylor University, Dept. of Surgery, Houston, Texas
DECAMP, PAUL T.---3503 Prytania St., New Orleans 15, La.
DELARUE, NORMAN C.---25 Donlea Drive, Toronto 17, Ont.
DENNIS, CLARENCE---Kryssarvägen 10, Näsby Park, Sweden

DESHAIES, GEORGES—37 Bellingham Road, Montreal, Que.
DETERLING, RALPH A., JR.—171 Harrison Ave., Boston 11, Mass.
DODRILL, FOREST D.—621 David Whitney Bldg., Detroit 26, Mich.
DOMM, SHELDON E.—1918 W. Clinch Ave., Knoxville 16, Tenn.
DORNER, RALPH A.—710 Equitable Bldg., Des Moines 9, Iowa
DORSEY, JOHN M.—636 Church St., Evanston, Ill.
DRAKE, EMERSON H.—18 Bramhall St., Portland 3, Maine
DRASH, EVERETT C.—University of Virginia Hospital, Charlottesville, Va.
DUGAN, DAVID J.—459 30th St., Oakland 9, Calif.

EFFLER, DONALD B.—Euclid and East 93rd Sts., Cleveland 6, Ohio
EHRENHAFT, JOHANN L.—University of Iowa, Iowa City, Iowa
ELLIS, F. HENRY, JR.—Mayo Clinic, Rochester, Minn.
ELLISON, ROBERT G.—Medical College of Georgia, Augusta, Ga.
EMERSON, GEORGE L.—11 Rochester St., Scottsville, N. Y.
EVANS, BYRON H.—2940 North Fresno St., Fresno 3, Calif.

FALOR, WILLIAM H.—623 Second National Bldg., Akron 8, Ohio
FELL, EGBERT H.—122 South Michigan Ave., Chicago 3, Ill.
FISCHER, WALTER W.—170 East 78th St., New York 21, N. Y.
FORD, WILLIAM B.—3500 Fifth Ave., Pittsburgh 13, Pa.
FORSEE, JAMES H., BRIG. GEN. (MC), USA
Walter Reed Army Medical Center, Washington 12, D. C.
FOX, ROBERT T.—2136 Robin Crest Lane, Glenview, Ill.
FRANK, HOWARD A.—330 Brookline Ave., Boston 15, Mass.
FRENCH, SANFORD W. III—Boron Community Hospital, Boron, Calif.

GAENSLER, EDWARD A.—229 Dudley Road, Newton Centre 59, Mass.
GAGNON, EDOUARD D.—902 Est., Rue Sherbrooke, Montreal, Que.
GEARY, PAUL—109 Park Ave., Plainfield, N. J.
GEBAUER, PAUL—Leahi Hospital, 649 Pokole St., Honolulu, Hawaii
GERBODE, FRANK—Presbyterian Medical Center, San Francisco 15, Calif.
GIBBON, JOHN H., JR.—1025 Walnut St., Philadelphia 7, Pa.
GLENN, FRANK—525 East 68th St., New York 21, N. Y.
GLENN, WM. W. L.—333 Cedar St., New Haven 4, Conn.
GLOVER, ROBERT P.—269 South 19th St., Philadelphia 3, Pa.
GOLDMAN, ALFRED—416 N. Bedford Drive, Beverly Hills, Calif.
GORDON, JOSEPH—717 Encino Plaza, N.E., Albuquerque, N. M.
GRACE, ARCHIBALD J.—530 Wellington St., London, Ont.
GRAVEL, JOFFRE-ANDRE—II Place George Vth, Quebec 4, Canada
GRIMES, ORVILLE F.—University of California Hospital, San Francisco 22, Calif.
GROSS, ROBERT E.—300 Longwood Ave., Boston, Mass.
GROVES, LAURENCE K.—Cleveland Clinic, Cleveland 6, Ohio
GROW, JOHN B.—3705 E. Colfax, Denver 6, Colo.

HAIGHT, CAMERON—University Hospital, Ann Arbor, Mich.
HANLON, ROLLINS—1325 S. Grand Blvd., St. Louis 4, Mo.
HARDY, JAMES D.—University of Mississippi Medical Center, Jackson, Miss.
HARKEN, DWIGHT E.—67 Bay State Road, Boston 15, Mass.
HARPER, FREDERICK R.—1825 Gilpin St., Denver 18, Colo.
HARRISON, ALBERT W.—Medical Branch, University of Texas, Galveston, Texas
HARRISON, ELLIOTT—1862 West Broadway, Vancouver 9, B. C.
HARTER, JOHN S.—212 Brown Bldg., Louisville 2, Ky.
HELMSWORTH, JAMES A.—Cincinnati General Hospital, Cincinnati 29, Ohio

HEROY, WILLIAM W.---East Gate Road, Lloyd Harbor, Huntington, N. Y.
HIGGINSON, JOHN F.---1430 Chapala St., Santa Barbara, Calif.
HOCHBERG, LEW A.---135 Eastern Parkway, Brooklyn 38, N. Y.
HOLINGER, PAUL H.---700 N. Michigan Ave., Chicago 11, Ill.
HOLMAN, CRANSTON W.---862 Fifth Ave., New York 21, N. Y.
HOPKINS, WILLIAM A.---1293 Peachtree St., N.E., Atlanta 9, Ga.
HUDSON, THEODORE R.---55 E. Washington St., Chicago 2, Ill.
HUFNAGEL, CHARLES A.---3800 Reservoir Road, N.W., Washington 7, D. C.
HUGHES, FELIX A., JR.---Kennedy Hospital, Memphis 17, Tenn.
HUMPHREYS, GEORGE H. II---180 Fort Washington Ave., New York 32, N. Y.
HURLEY, GERARD A. P.---1538 Sherbrooke St., W., Montreal, Que.
HURWITT, ELLIOTT S.---Montefiore Hospital, New York 67, N. Y.
HURWITZ, ALFRED---Maimonides Hospital, 4802 Tenth Ave., Brooklyn 19, N. Y.

JARVIS, FRED J.---1115 Columbia St., Seattle 4, Wash.
JOHNSON, ELGIE K.---230 Hilton St., Hempstead, N. Y.
JOHNSON, JULIAN---3400 Spruce St., Philadelphia 4, Pa.
JOHNSTON, JAMES H., JR.---710 N. State St., Jackson 2, Miss.
JONES, JOHN C.---1136 West 6th St., Los Angeles 17, Calif.
JOYNT, G. HARRY C.---399 Bathurst St., Toronto, Ont.
JULIAN, ORMAND C.---25 E. Washington St., Chicago 2, Ill.

KARLSON, KARL E.---451 Clarkson Ave., Brooklyn 3, N. Y.
KAUSEL, HARVEY W.---Albany Hospital, Albany 8, N. Y.
KAY, EARLE B.---10465 Carnegie Ave., Cleveland 6, Ohio
KEE, JOHN LESTER---3810 Swiss Ave., Dallas, Texas
KEELEY, JOHN L.---30 North Michigan Ave., Chicago 2, Ill.
KELLEY, WINFIELD O.---Uncas-on-Thames, Norwich, Conn.
KENT, EDWARD M.---3500 Fifth Ave., Pittsburgh 13, Pa.
KERGIN, FREDERICK G.---139 Private Patients Pavilion, Toronto General Hosp., Toronto 2, Ont.
KESSLER, CHARLES R.---1321 21st Way, South, Birmingham 5, Ala.
KEY, JAMES A.---170 St. George St., Toronto, Ont.
KING, RICHARD---Suite 233, 340 Boulevard, N.E., Atlanta 12, Ga.
KINSELLA, THOMAS J.---1251 Medical Arts Bldg., Minneapolis 2, Minn.
KIRBY, CHARLES K.---3400 Spruce St., Philadelphia 4, Pa.
KIRKLIN, JOHN W.---Mayo Clinic, Rochester, Minn.
KIRSCHNER, PAUL A.---2 East 92nd St., New York 28, N. Y.
KITTLE, C. FREDERICK---University of Kansas Medical Center, Kansas City 12, Kan.
KLASSEN, KARL P.---Ohio State University, Columbus 15, Ohio
KLEPSER, ROY G.---1835 Eye St., N.W., Washington 6, D. C.
KLOPSTOCK, ROBERT---Veterans Adm. Hospital, Brooklyn 9, N. Y.

LAIRD, ROBERT---399 Bathurst St., Toronto, Ont.
LAM, CONRAD R.---Henry Ford Hospital, Detroit 2, Mich.
LAMBERT, ADRAIN---768 Park Ave., New York 21, N. Y.
LAMPSON, R. STARR---85 Jefferson St., Hartford 14, Conn.
LANGSTON, HIRAM T.---1919 West Taylor St., Chicago 12, Ill.
LAUREY, JAMES R.---5710 16th St., N.W., Washington 11, D. C.
LEEDS, SANFORD E.---2211 Post St., San Francisco 15, Calif.
LEES, WILLIAM M.---7000 N. Kenton Ave., Lincolnwood 46, Ill.
LEWIS, F. JOHN---Northwestern University Medical School, Chicago 11, Ill.
LILLEHEI, C. WALTON---University Hospitals, Minneapolis 14, Minn.
LINDSKOG, GUSTAF E.---50 Marvel Road, New Haven, Conn.
LONGMIRE, WILLIAM P., JR.---UCLA School of Medicine, Los Angeles 24, Calif.
LYNCH, JOSEPH P.---1180 Beacon St., Brookline 46, Mass.

MACKLER, S. ALLEN—104 S. Michigan Ave., Chicago 3, Ill.
MACMANUS, JOSEPH—491 Delaware, Buffalo 2, N. Y.
MAHONEY, EARLE B.—260 Crittenden Blvd., Rochester 20, N. Y.
MAIER, HERBERT C.—3 East 71st St., New York 21, N. Y.
MAJOR, ROBERT C.—1477 Harper St., Augusta, Ga.
MANNIX, EDGAR P., JR.—12 Forest Turn, Manhasset, Long Island, N. Y.
MAURER, ELMER P. R.—827 Union Central Bldg., Cincinnati 2, Ohio
MAUTZ, F. R.—10515 Carnegie Ave., Cleveland 6, Ohio
MAYER, JOHN H., JR.—503 Plaza Parkway Bldg., Kansas City 12, Mo.
MCDONALD, JOHN R.—Harper Hospital, 3825 Brush St., Detroit, Mich.
MELTZER, HERBERT—505 Medical Arts Bldg., Edmonton, Alberta, Canada
MENDELSOHN, HARVEY J.—2065 Adelbert Road, Cleveland 6, Ohio
MERENDINO, K. ALVIN—University of Washington, Seattle 5, Wash.
MERKEL, CARL G.—8 Church St., Saranac Lake, N. Y.
MEYER, BERTRAND W.—922 Keatley Road, La Canada, Calif.
MICHELSON, ELLIOTT—1801 Eutaw Place, Baltimore 17, Md.
MILLS, WALDO O.—1445 Medical and Dental Bldg., Seattle 1, Wash.
MINOR, GEORGE R.—University of Virginia Hospital, Charlottesville, Va.
MISCALL, LAURENCE—11 East 68th St., New York, N. Y.
MORROW, ANDREW G.—National Heart Institute, Bethesda 14, Md.
MOULDER, PETER V.—950 East 59th St., Chicago 37, Ill.
MULLER, WM. H., JR.—University of Virginia Hospital, Charlottesville, Va.
MULVIHILL, DANIEL A.—15 East 77th St., New York 21, N. Y.

NEALON, THOMAS F., JR.—1025 Walnut St., Philadelphia 7, Pa.
NEMIR, PAUL, JR.—University of Pennsylvania, Philadelphia 4, Pa.
NEPTUNE, WILFORD B.—135 Francis St., Boston 15, Mass.
NEWMAN, MELVIN M.—3800 E. Colfax Ave., Denver 6, Colo.

OATWAY, WILLIAM H., JR.—La Vina Sanatorium, Altadena, Calif.
OLSEN, ARTHUR M.—102 2nd Ave., S.W., Rochester, Minn.
O'NEILL, THOMAS J. E.—Suite 110 Centennial Bldg., Philadelphia 25, Pa.
O'ROURKE, PAUL V.—307 David Whitney Bldg., Detroit 26, Mich.
OVERHOLT, RICHARD H.—135 Francis St., Boston 15, Mass.

PAINE, JOHN R.—Buffalo General Hospital, 100 High St., Buffalo 14, N. Y.
PAPPER, EMANUEL M.—622 West 168th St., New York 32, N. Y.
PARKER, EDWARD F.—158 Rutledge Ave., Charleston 8, S. C.
PAULSON, DONALD L.—3810 Swiss Ave., Dallas, Texas
PETERS, RICHARD M.—University of North Carolina, Chapel Hill, N. C.
PHILLIPS, FRANCIS J.—1016 Fireweed Lane, Anchorage, Alaska
POOL, JOHN L.—755 Park Ave., New York 21, N. Y.
POPPE, J. KARL—1130 S.W. Morrison St., Portland 5, Ore.
POTTS, WILLIS J.—707 Fullerton Ave., Chicago 14, Ill.

RAMSEY, BEATTY H.—2210 Santa Monica Blvd., Santa Monica, Calif.
RASMUSSEN, RICHARD A.—Blodgett Medical Bldg., Grand Rapids 6, Mich.
RAVITCH, MARK M.—Johns Hopkins Hospital, Baltimore, 5, Md.
READ, CHARLES T.—550 West Thomas Road, Phoenix, Ariz.
RICHARDS, VICTOR—Stanford-Lane Hospital, San Francisco 15, Calif.
RIGGINS, H. MCLEOD—1031 Fifth Ave., New York 28, N. Y.
RIPSTEIN, CHARLES B.—15 Birch St., Great Neck, L. I., N. Y.
ROBERTSON, ROSS—925 West Georgia St., Vancouver 1, B. C.
ROE, BENSON B.—University of California Medical Center, San Francisco 22, Calif.

ROGERS, W. L.----490 Post St., San Francisco 2, Calif.
ROSEMOND, GEORGE P.----3401 Broad St., N., Philadelphia 40, Pa.
RUMEL, WILLIAM R.----535 East 1st South, Salt Lake City 2, Utah

SABISTON, DAVID C.----Johns Hopkins Hospital, Baltimore 5, Md.
SALYER, JOHN M.----2032 North Broadway, Santa Ana, Calif.
SAMSON, PAUL C.----3959 Happy Valley Road, Lafayette, Calif.
SANGER, PAUL W.----Doctors Bldg., Kings Drive, Charlotte, N. C.
SAROT, IRVING A.----111 East 69th St., New York 21, N. Y.
SCANNELL, J. GORDON----Massachusetts General Hospital, Boston 14, Mass.
SCHAFFNER, VERNON D.----12 Cornwallis St., Kentville, Nova Scotia
SCHMIDT, HERBERT WM.----Mayo Clinic, Rochester, Minn.
SCOTT, HENRY W., JR.----Vanderbilt University Hospital, Nashville 5, Tenn.
SEALY, WILL C.----Duke University Hospital, Durham, N. C.
SEILER, HAWLEY H.----517 Bayshore Blvd., Tampa 6, Fla.
SELEY, GABRIEL P.----540 Park Ave., New York 21, N. Y.
SHAW, ROBERT R.----3810 Swiss Ave., Dallas, Texas
SHEFTS, LAWRENCE M.----614 Medical Professional Bldg., San Antonio 12, Texas
SHUMACKER, HARRIS B., JR.----Indiana University Medical Center, Indianapolis 7, Ind.
SKINNER, EDWARD F.----20 S. Dudley St., Memphis 3, Tenn.
SLOAN, HERBERT----University Hospital, Ann Arbor, Mich.
SNYDER, JOHN M.----1236 Moffitt Ave., Bethlehem, Pa.
SOMMER, GEORGE N. J., JR.----120 W. State St., Trenton 8, N. J.
SOUTTER, LAMAR----80 East Concord St., Boston 18, Mass.
SPENCER, FRANK C.----Johns Hopkins Hospital, Baltimore 5, Md.
STARKEY, GEORGE W. B.----319 Longwood Ave., Boston 15, Mass.
STEELE, J. D.----Veterans Adm. Hospital, San Fernando, Calif.
STEPHENS, H. BRODIE----384 Post St., San Francisco 8, Calif.
STOREY, CLIFFORD F.----1309 Medical-Dental Bldg., San Diego 1, Calif.
STRANAHAN, ALLEN----Albany Hospital, Albany, N. Y.
STRIEDER, JOHN W.----1180 Beacon St., Brookline 46, Mass.
STRODE, JOSEPH E.----Straub Clinic, Kapiolani St. at Thomas Square, Honolulu 14, Hawaii
STRUG, LAWRENCE H.----2419 Jefferson Ave., New Orleans 15, La.
SWAN, HENRY II----4200 East 9th Ave., Denver 20, Colo.
SWEET, RICHARD H.----87 Chestnut St., Boston 8, Mass.

TABER, RODMAN E.----Henry Ford Hospital, Detroit 2, Mich.
TAYLOR, FREDERICK H.----1012. Kings Drive, Charlotte, N. C.
TEMPLETON, JOHN Y. III----1025 Walnut St., Philadelphia 7, Pa.
THOMAS, GORDON W.----Int. Grenfell Association, St. Anthony, Newfoundland
TUTTLE, WILLIAM M.----307 David Whitney Bldg., Detroit 26, Mich.

VAN HAZEL, WILLARD----224 S. Michigan Blvd., Chicago 4, Ill.
VARCO, RICHARD L.----University Hospitals, Minneapolis 14, Minn.
VINEBERG, ARTHUR M.----1390 Sherbrooke St., W., Montreal 25, Que.
VORWALD, ARTHUR J.----College of Medicine, Wayne State University, Detroit 7, Mich.

WADDELL, WILLIAM R.----69 Woodland Road, Chestnut Hill, Mass.
WALKER, JAMES H.----1323 Quarrier St., East, Charleston 1, W. Va.
WALKUP, HARRY E.----Veterans Adm. Hospital, Oteen, N. C.
WARE, PAUL F.----124 Russell St., Worcester, Mass.
WATERMAN, DAVID H.----1918 West Clinch Ave., Knoxville 16, Tenn.
WATKINS, ELTON, JR.----Lahey Clinic, 605 Commonwealth Ave., Boston 15, Mass.
WATSON, WILLIAM L.----340 East 72nd St., New York 21, N. Y.

WEBB, WATTS R.—University Hospital, Jackson, Miss.
WEISEL, WILSON—324 E. Wisconsin Ave., Milwaukee 2, Wis.
WHITE, MARION L., JR.—Huntington Bank Bldg., Huntington, W. Va.
WILLIAMS, MARK H.—63 Front St., Binghamton, N. Y.
WILSON, NORMAN J.—135 Francis St., Boston 15, Mass.
WIPER, THOMAS B.—Suite 1336, 450 Sutter St., San Francisco 8, Calif.
WOLFF, WILLIAM I.—30 Central Park South, New York 19, N. Y.
WOODRUFF, WARRINER—8 Church St., Saranac Lake, N. Y.
WOODS, FRANCIS M.—135 Francis St., Boston 15, Mass.
WRIGHT, GEORGE W.—11311 Shaker Blvd., Cleveland 4, Ohio
WYLIE, ROBERT H.—903 Park Ave., New York, N. Y.

Associate Members

ACKMAN, F. DOUGLAS—1374 Sherbrooke St., W., Montreal 25, Que.
ADAMS, JESSE E., JR.—301 Medical Arts Bldg., Chattanooga 2, Tenn.
ADELMAN, ARTHUR—751 East 63rd St., Kansas City 10, Mo.
ADKINS, PAUL C.—901 Twenty-third St., N.W., Washington 7, D. C.
AITCHISON, DAVID B.—Mountain Sanatorium, Hamilton, Ont.
ANDERSEN, MURRAY N.—462 Grider St., Buffalo 15, N. Y.
ANKENEY, JAY L.—2065 Adelbert Road, Cleveland 6, Ohio

BARRETT, RAYMOND J.—18280 Fairfield St., Detroit 21, Mich.
BENOIT, HECTOR W., JR.—503 Plaza Parkway Bldg., Kansas City 12, Mo.
BERGMANN, MARTIN—4500 Olive St., St. Louis 8, Mo.
BESKIN, CHARLES A.—3920 Convention St., Baton Rouge, La.
BLALOCK, JOHN B.—3503 Prytania St., New Orleans 15, La.
BLOOMBERG, ALLAN E.—1095 Park Ave., New York 28, N. Y.
BLOOMER, WILLIAM E.—333 Cedar St., New Haven 4, Conn.
BOUSQUET, ERNEST O.—5689 Boulevard Rosemont, Montreal, Que.
BROOKS, JAMES W.—1200 E. Broad St., Richmond 19, Va.
BRYANT, JOSEPH R.—321 West Broadway, Louisville 2, Ky.
BURBANK, BENJAMIN—244 Henry St., Brooklyn 1, N. Y.

CAHAN, WILLIAM G.—444 E. 68th St., New York 21, N. Y.
CAMPBELL, GILBERT S.—612 N.E. 17th St., Oklahoma City, Okla.
CHANDLER, JOHN H.—The Jackson Clinic, 616 W. Forest Ave., Jackson, Tenn.
CHODOFF, RICHARD J.—255 South 17th St., Philadelphia 3, Pa.
CHUNN, CHARLES F.—442 W. Lafayette St., Tampa 6, Fla.
CINCOTTI, JOHN J.—Veterans Adm. Hospital, Albany, N. Y.
COLE, FRANCIS H.—1375 Goodbar Ave., Memphis, Tenn.
CONNAR, RICHARD G.—706 Franklin St., Tampa 2, Fla.
CONNOLLY, JOHN E.—Presbyterian Medical Center, San Francisco 15, Calif.
COOKE, FRANCIS N.—25 S.E. Second Ave., Miami 32, Fla.
COWLEY, R. ADAMS—University Hospital, Baltimore 1, Md.
COX, WILLIAM V.—133 Court St., Auburn, Maine
CRACOVANER, ARTHUR J.—103 East 78th St., New York 21, N. Y.
CRASTNOPOL, PHILIP—1221 East 21st St., Brooklyn 10, N. Y.
CRECCA, ANTHONY D.—376 Roseville Ave., Newark 7, N. J.

DAFOE, COLIN S.—508 Medical Arts Bldg., Edmonton, Alberta
DASCH, FREDERICK W.—416 West Market St., Pottsville, Pa.
DAVIS, MILTON V.—3707 Gaston Ave., Dallas 10, Texas
DEATON, W. RALPH, JR.—1027 Professional Village, Greensboro, N. C.

DEBORD, ROBERT A.---1240 Jefferson Bldg., Peoria, Ill.
DECKER, ALFRED M., JR.---8 Church St., Saranac Lake, N. Y.
DEMATTEIS, ALBERT---2612 Pleasant Valley Blvd., Altoona, Pa.
DESFORGES, GERARD---49 Lake Ave., Melrose 76, Mass.
DIVELEY, WALTER L.---410 Medical Arts Bldg., Nashville 12, Tenn.
DODDS, G. ALFRED---807 Broadway, Fargo, N. D.

FERGUSON, THOMAS B.---Barnes Hospital, St. Louis 10, Mo.
FINDLAY, CHARLES W., JR.---180 Fort Washington Ave., New York 32, N. Y.
FINNERTY, JAMES---Brookhaven Medical Arts Bldg., Patchogue, N. Y.
FISHBACK, FREDERICK C.---1835 Eye St., N.W., Washington 6, D. C.
FITZPATRICK, HUGH F.---St. Luke's Hospital, New York 25, N. Y.
FORD, JOSEPH M.---1056 Fifth Ave., New York 28, N. Y.
FRIEDLANDER, RALPH---The Bronx Hospital, New York 56, N. Y.
FRIESEN, STANLEY R.---39th and Rainbow, Kansas City 3, Kan.
FULLER, JOSIAH---205 W. 2nd St., Duluth 2, Minn.

GARAMELLA, JOSEPH J.---1629 Medical Arts Bldg., Minneapolis, Minn.
GERBASI, FRANCIS S.---Alexander Blain Hospital & Clinic, Detroit 7, Mich.
GREER, ALLEN E.---1200 North Walker, Oklahoma City, Okla.
GWATHMEY, OWEN---501 East Franklin St., Richmond 19, Va.

HAMPTON, FOSTER, JR.---Suite 101, Interstate Bldg., Chattanooga, Tenn.
HANNER, JOSEPH M.---U. S. Naval Hospital, San Diego 34, Calif.
HAUSMANN, PAUL F.---2309 West State St., Milwaukee 3, Wis.
HEANEY, JOHN P.---1608 Nix Professional Bldg., San Antonio 5, Texas
HERRERA, RODOLFO---11 Calle #2-37, Guatemala City 1, Guatemala
HERTZLER, JACK H.---4377 West Maple Road, Birmingham, Mich.
HEWLETT, THOMAS H.---Brooke Army Hospital, Fort Sam Houston, Texas
HILL, LUCIUS D. III---1118 Ninth Ave., Seattle 1, Wash.
HOLLAND, ROBERT H.---620 Glen Arbor Court, Dallas 16, Texas
HOOD, R. MAURICE---806 Capital National Bank Bldg., Austin 16, Texas
HUN, HENRY H.---149 Washington Ave., Albany 10, N. Y.

INGRAM, IVAN N.---655 Sutter St., San Francisco 2, Calif.
IOVINE, VINCENT M.---1150 Connecticut Ave., N.W., Washington 6, D. C.

JAHNKE, EDWARD J., JR.---97th General Hospital, APO 757, New York, N. Y.
JAMPLIS, ROBERT W.---300 Homer Ave., Palo Alto, Calif.
JENSEN, NATHAN K.---1629 Medical Arts Bldg., Minneapolis 2, Minn.
JENSIK, ROBERT J.---224 S. Michigan Ave., Chicago 4, Ill.
JOHNS, THOMAS N. P.---6305 Towana Road, Richmond 13, Va.
JOHNSON, CLIVE R.---800 Fifth Ave., Fort Worth 4, Texas
JOHNSON, FRANK E.---829 Medical Arts Bldg., Minneapolis 2, Minn.
JOHNSTON, FRANK R.---Bowman Gray School of Medicine, Winston-Salem, N. C.
JUDD, ARCHIBALD R.---304 N. Fourth St., Hamburg, Pa.

KAUNITZ, VICTOR H.---4083 Delaware Ave., Tonawanda, N. Y.
KAY, JEROME HAROLD---122 North Alta Vista Blvd., Los Angeles 36, Calif.
KEMLER, R. LEONARD---576 Farmington Ave., Hartford 5, Conn.
KENNEY, LEO J.---610 Medical Arts Bldg., Grand Rapids 2, Mich.
KRAEFT, NELSON H.---1501 Magnolia Drive, Tallahassee, Fla.
KUNDERMAN, PHILIP J.---185 Livingston Ave., New Brunswick, N. J.
KUNSTLER, WALTER E.---1538 Sherbrooke St., W., Montreal 25, Que.

LAWRENCE, GEORGE HUGH—1118 Ninth Ave., Seattle 1, Wash.
LEIBOVITZ, MARTIN—812 Medical Arts Bldg., Tulsa 3, Okla.
LEWIS, J. EUGENE, JR.—634 North Grand Blvd., St. Louis 3, Mo.
LEWIS, RUBIN M.—2380 Ellsworth, Berkeley 4, Calif.
LITTLEFIELD, JAMES B.—University of Virginia Hospital, Charlottesville, Va.
LUCIDO, JOSEPH L.—634 North Grand Blvd., St. Louis 3, Mo.
LYNN, R. BEVERLEY—R. R. #1, Westbrook, Ontario, Canada
LYON, CLAYTON—384 Post St., San Francisco 8, Calif.

MACDONALD, NEIL—Medical Arts Bldg., Windsor, Ont.
MACPHERSON, LACHLAN—St. John Tuberculosis Hospital, East St. John, N. B.
MADOFF, IRVING M.—1180 Beacon St., Brookline 46, Mass.
MAHAFFEY, DANIEL E.—1112 Heyburn Bldg., Louisville 2, Ky.
MALONEY, JAMES V., JR.—UCLA School of Medicine, Los Angeles 24, Calif.
MANGIARDI, JOSEPH L.—520 Franklin Ave., Garden City, N. Y.
MASON, JAMES M. III—1023 South 20th St., Birmingham 5, Ala.
MCBURNEY, ROBERT P.—899 Madison Ave., Memphis 3, Tenn.
MCGOON, DWIGHT C.—Mayo Clinic, Rochester, Minn.
MECKSTROTH, CHARLES V.—University Hospital, Columbus 10, Ohio
MELICK, DERMONT W.—1005 Professional Bldg., Phoenix, Ariz.
MENDELSSOHN, EDWIN—1301 Tabor Road, Philadelphia, Pa.
MILLER, ARTHUR C.—Loma Linda Medical Center, Loma Linda, Calif.
MILLER, CARROLL C.—304 Humphrey St., Swampscott, Mass.
MILLER, FLETCHER A.—Mount Sinai Hospital, Minneapolis 4, Minn.
MOORE, THOMAS C.—Indiana University Medical Center, Indianapolis, Ind.
MORRIS, JOE D.—1801 Weldon Blvd., Ann Arbor, Mich.
MOUSEL, LLOYD H.—Dept. of Anesthesiology, The Swedish Hospital, Seattle 4, Wash.
MUNNELL, EDWARD R.—301 N.W. 12th St., Oklahoma City 3, Okla.
MUSTARD, WILLIAM T.—200 St. Clair Ave., W., Toronto 7, Ont.

NARDI, GEORGE L.—Massachusetts General Hospital, Boston 14, Mass.
NEERKEN, ADRIAN J.—1318 American National Bank Bldg., Kalamazoo 4, Mich.
NELSON, RUSSELL M.—325 Eighth Ave., Salt Lake City 3, Utah
NETTERVILLE, RUSH E.—514 E. Woodrow Wilson Drive, Jackson 6, Miss.
NEWMAN, ROBERT W.—Medical Arts Bldg., Knoxville, Tenn.

O'NEILL, JAMES F.—140 Roslyn Ave., Glenside, Pa.
OVERSTREET, JOHN WM.—508 Hermann Professional Bldg., Houston 25, Texas

PAUL, JOHN S.—Baker VA Center, Martinsburg, W. Va.
PEABODY, JOSEPH W., JR.—1150 Connecticut Ave., N.W., Washington 6, D. C.
PECORA, DAVID V.—Box 20, Ray Brook, N. Y.
PERKINS, REX BEACH—1919 Seventh Ave., South, Birmingham 3, Ala.
PINKHAM, ROLAND D.—Suite 1445, Medical & Dental Bldg., Seattle 1, Wash.
POLK, JOHN W.—315 Professional Bldg., Springfield 4, Mo.
PONTIUS, ROBERT G.—125 DeSoto St., Pittsburgh 13, Pa.
PRATT, LAWRENCE A.—15621 Windmill Pt., Grosse Pointe Park 30, Mich.

QUINLAN, JOHN J.—Nova Scotia Sanatorium, Kentville, Nova Scotia

RANSDELL, HERBERT T., JR.—Louisville General Hospital, Louisville 2, Ky.
ROBBINS, S. GWIN—899 Madison Ave., Memphis 3, Tenn.
ROBINSON, GEORGE—20 Archer Ave., Mount Vernon, N. Y.
ROBINSON, JOSEPH L.—1136 West Sixth St., Los Angeles 17, Calif.
ROSENBERG, DENNIS M.—1539 Delachaise St., New Orleans 15, La.

ROSS, RALEIGH R.—2 Medical Arts Square, Austin 5, Texas
RUBENSTEIN, LAURENCE H.—571 Woodlawn Ave., Glencoe, Ill.
RUBIN, MORRIS—2021 Grand Concourse, Bronx 53, N. Y.
RYAN, BERNARD J.—375 East Main St., Bay Shore, N. Y.
RYAN, THOMAS C.—90 Shenango St., Greenville, Pa.

SANES, GILMORE M.—3500 Fifth Ave., Pittsburgh 13, Pa.
SCHRAMEL, ROBERT J.—1430 Tulane Ave., New Orleans 12, La.
SELMAN, MORRIS W.—2302 Meadowwood Drive, Toledo 2, Ohio
SEYBOLD, WILLIAM D.—Hermann Professional Bldg., Houston 25, Texas
SHIELDS, THOMAS W.—700 North Michigan Ave., Chicago 11, Ill.
SIRAK, HOWARD D.—Ohio State University Hospital, Columbus 10, Ohio
SKINNER, A. M.—Home Folks Tuberculosis Hospital, Oneonta, N. Y.
SNYDER, HOWARD E.—103½ E. Ninth Ave., Winfield, Kan.
SPEAR, HAROLD C.—1550 N.W. 10th Ave., Miami 36, Fla.
STATE, DAVID—Albert Einstein College of Medicine, New York 61, N. Y.
STAYMAN, JOSEPH W.—8815 Germantown Ave., Philadelphia 18, Pa.
STENSTROM, JOHN D.—220-1105 Pandora Ave., Victoria, B. C.
SULLIVAN, HERBERT J.—Medical Arts Bldg., Hamilton, Ont.
SWENSON, ORVAR—20 Ash St., Boston 11, Mass.

TAKARO, TIMOTHY—Veterans Adm. Hospital, Oteen, N. C.
TAYLOR, WARREN J.—67 Bay State Road, Boston 15, Mass.
TEST, FREDERICK C. II—20252 Meyers Road, Detroit 35, Mich.
TILLOU, DONALD J.—311 W. Church St., Elmira, N. Y.
TIMMES, JOSEPH J.—U. S. Naval Hospital, St. Albans 14, N. Y.
TOCKER, ALFRED M.—2110 E. Douglas, Wichita 14, Kan.
TRICERRI, FERNANDO E.—3 Chemin Mornex, Lausanne, Switzerland

VALLE, A. R.—U.S.P.H.S. Hospital, Detroit 15, Mich.
VAN FLEIT, WILLIAM E.—Emory University Clinic, Atlanta 22, Ga.
WALKER, GEORGE R.—555 University Ave., Toronto, Ont., Canada
WATKINS, DAVID H.—Denver General Hospital, Denver 4, Colo.
WEINBERG, MILTON, JR.—2632 Central Park Ave., Evanston, Ill.
WHITESIDE, WILLIAM C.—415 Medical Arts Bldg., Victoria, B. C.
WILKINS, EARLE W., JR.—275 Charles St., Boston 14, Mass.
WILSON, JOHN L.—Dept. Surgery, American University of Beirut, Beirut, Lebanon
WITMER, ROBERT H.—126 East Chestnut St., Lancaster, Pa.
WOLCOTT, MARK W.—Veterans Adm. Hospital, Coral Gables, Fla.

YOUNG, WILLIAM GLENN, JR.—Duke University Medical Center, Box 3617, Durham, N. C.

Senior Members

AMBERSON, J. B.—Bellevue Hospital, New York 16, N. Y.
AUFSES, ARTHUR H.—812 Park Ave., New York 21, N. Y.

BADGER, THEODORE L.—264 Beacon St., Boston 16, Mass.
BALLON, DAVID H.—1538 Sherbrooke St., N., Montreal 25, Que.
BARNWELL, JOHN B.—Room 890F, Veterans Administration, Washington 25, D. C.
BECK, CLAUDE S.—2065 Adelbert Road, Cleveland 6, Ohio
BENEDICT, EDWARD B.—Massachusetts General Hospital, Boston 14, Mass.
BERRY, FRANK B.—4301 Massachusetts Ave., Washington 16, D. C.
BETTMAN, RALPH B.—104 South Michigan Ave., Chicago, Ill.

BETTS, REEVE H.—Christian Medical College, Vellore, South India
BIRD, CLARENCE E.—64 Alfred Stone Rd., Providence 6, R. I.
BISGARD, J. DEWEY—422 Doctors Bldg., Omaha 31, Neb.
BLALOCK, ALFRED—Johns Hopkins Hospital, Baltimore 5, Md.
BLOCH, ROBERT G.—Montefiore Hospital, New York 67, N. Y.
BROWN, A. LINCOLN—490 Post St., San Francisco 2, Calif.
BURNETT, W. EMORY—Broad and Ontario Streets, Philadelphia 40, Pa.
BUTLER, ETHAN FLAGG—956 West Water St., Elmira, N. Y.
BYERS, H. RODDICK—Ganonoque, Ont.

CARLSON, HERBERT A.—4241 East 14th St., Long Beach, Calif.
CARTER, B. NOLAND—Madeira, Cincinnati 43, Ohio
CHURCHILL, EDWARD D.—Massachusetts General Hospital, Boston 14, Mass.
CLERF, LOUIS H.—5575 Eighth Ave., North, St. Petersburg 2, Fla.
COLE, DEAN B.—Professional Bldg., Richmond, Va.
COOPER, DAVID A.—1520 Spruce St., Philadelphia 2, Pa.
COURNAND, ANDRE—Bellevue Hospital, 27th St. & 1st Ave., New York 16, N. Y.
CURTIS, GEORGE M.—Ohio State University College of Medicine, Columbus, Ohio

DAVIDSON, LOUIS R.—30 East 60th St., New York 22, N. Y.
DECKER, HARRY R.—730 The Park Bldg., 355 5th Ave., Pittsburgh 22, Pa.
DOLLEY, FRANK S.—2010 Wilshire Blvd., Los Angeles 57, Calif.
DOUGLASS, RICHMOND—Veterans Adm. Hospital, Castle Point, N. Y.
DOVELL, CHAUNCEY, COL. (MC), USA (RET.)—62 South Boxwood St., Hampton, Va.

ELOESSER, LEO—490 Post St., San Francisco 2, Calif.

FAULKNER, WILLIAM B., JR.—20 San Rafael Way, San Francisco 27, Calif.
FERGUSON, R. G.—Balfour Apts., Regina, Sask.
FLICK, JOHN B.—1617 Pennsylvania Blvd., Philadelphia 3, Pa.
FREELANDER, SAMUEL O.—2460 Fairmount Blvd., Cleveland Heights 6, Ohio

GALE, JOSEPH W.—University Hospitals, Madison 6, Wis.
GARLOCK, JOHN H.—47 East 77th St., New York 21, N. Y.

HARRINGTON, STUART W.—Mayo Clinic, Rochester, Minn.
HARRISON, HARLON W., CAPT. (MC), USNR—U. S. Naval Hospital, San Diego, Calif.
HART, DERYL—Duke University, Durham, N. C.
HAYES, JOHN N.—24 Church St., Saranac Lake, N. Y.
HEAD, JEROME R.—55 E. Washington St., Chicago 2, Ill.
HEINBECKER, PETER—Washington University Medical School, St. Louis 10, Mo.
HOLMAN, EMILE—Presbyterian Medical Center, Clay & Webster Sts., San Francisco 15, Calif.
HUDSON, WILLIAM A.—602 David Whitney Bldg., Detroit, Mich.

JACKSON, CHEVALIER L.—3401 N. Broad St., Philadelphia 40, Pa.
JANES, ERNEST C.—250 Main St., East, Hamilton, Ontario
JANES, ROBERT M.—904 Medical Arts Bldg., Toronto 5, Ont.
JOHNS, FRANK S.—Johnston-Willis Hospital, Richmond 21, Va.
JOHNSON, HOLLIS E.—2122 West End Ave., Nashville 5, Tenn.

KERNAN, JOHN D.—103 East 78th St., New York, N. Y.
KING, DONALD S.—Hitchcock Clinic, Hanover, N. H.
KIPP, HAROLD A.—Mercy Hospital, Pittsburgh 15, Pa.
KNOEPP, LOUIS F.—Veterans Adm. Hospital, Alexandria, La.

LEAHY, LEON J.---105 Medical Arts Bldg., Buffalo 2, N. Y.
LESTER, CHARLES W.---70 E. 80th St., New York 21, N. Y.
LEVEN, N. LOGAN---1464 Lowry Medical Arts Bldg., St. Paul 2, Minn.
LEWALD, LEON T.---1200 Fifth Ave., New York 29, N. Y.
LOCKWOOD, A. L.---300 Bloor St., E., Toronto, Ont.

MCINTOSH, CLARENCE A.---1390 Sherbrooke St., W., Montreal, Que.
MEADE, RICHARD H.---750 San Jose Drive, S.E., Grand Rapids, Mich.
MEYER, HERBERT WILLY---Box 507, Rancho Santa Fe, Calif.
MILLER, ROBERT T., JR.---Mountain Lake, Lake Wales, Fla.
MOERSCH, HERMAN---1064 Plummer Lane, Rochester, Minn.
MOORE, RICHMOND L.---180 Ft. Washington Ave., New York 32, N. Y.
MUDD, JAMES L.---634 N. Grand Blvd., St. Louis 3, Mo.
MURPHY, JAMES D.--- ---U. S. Veterans Adm. Hospital, Baltimore 18, Md.
MYERS, J. ARTHUR---730 La Salle Bldg., Minneapolis, Minn.

NEUHOF, HAROLD---Box 198, Huntington Road, Stratford, Conn.
NIXON, JAMES W.---1121 Nix Professional Bldg., San Antonio 5, Texas

OCHSNER, ALTON---Ochsner Clinic, 3503 Prytania St., New Orleans 15, La.
ORNSTEIN, GEORGE---965 Fifth Ave., New York, N. Y.

PACKARD, EDWARD N.---142 Park Ave., Saranac Lake, N. Y.
PICKHARDT, OTTO C.---66 East 79th St., New York, N. Y.
PROCTER, OSCAR S.---Box 1053, Topeka, Kan.

RAINE, FORRESTER---2230 E. Bradford Ave., Milwaukee 11, Wis.
RIENHOFF, WILLIAM F., JR.---1201 N. Calvert St., Baltimore 2, Md.
RIGLER, LEO G.---Cedars of Lebanon Hospital, Los Angeles 29, Calif.
ROSS, DUDLEY E.---St. Adolphe de Howard, P.Q., Canada

SKINNER, GEORGE F.---36 Coburg St., St. John, New Brunswick
SMITH, DAVID T.---Duke University, Durham, N. C.

THOMPSON, SAMUEL A.---850 Park Ave., New York 21, N. Y.
THORBURN, GRANT---1602 West Genessee St., Flint, Mich.
TOUROFF, ARTHUR S. W.---994 Fifth Ave., New York 28, N. Y.
TYSON, M. DAWSON---Hitchcock Clinic, Hanover, N. H.

VAN ALLEN, CHESTER M.---State Hospital, Bikaner, Rajputana, India

WANGENSTEEN, OWEN H.---University Hospitals, Minneapolis 14, Minn.
WEINBERG, JOSEPH A.---Veterans Adm. Hospital, Long Beach 4, Calif.
WELLES, EDWARD S.---20 Church St., Saranac Lake, N. Y.
WILLAUER, GEORGE---1930 Chestnut St., Philadelphia, Pa.
WILSON, JULIUS LANE---1790 Broadway, New York 19, N. Y.

Members Deceased

A. T. BAZIN
RICHARD H. DIEFFENBACH
AARON HIMMELSTEIN
WILLIAM L. KELLER
VICTOR O. MADER
JULIAN A. MOORE
EDWARD J. O'BRIEN

ANNUAL MEETING NOTES

The Fortieth Annual Meeting of The American Association for Thoracic Surgery was held in Miami Beach, Florida, May 11, 12, and 13, 1960. There were 892 physicians who registered for the scientific sessions. In addition there were 109 wives present. The representation from foreign countries was rather impressive, particularly from the Far East.

The meeting arrangements were satisfactory except for an unavoidable and incomprehensible mismanagement of the hotel reservation arrangements at The Deauville, as well as their satellite and supporting institutions along the Beach.

As a result of extended deliberations over several years, by the Council, by special committees, as well as the Executive Session and the voting membership, the following proposals affecting the membership structure of the Association and embodying changes in the Constitution as well as the By-Laws were adopted after due process.

CONSTITUTION: ARTICLE III. MEMBERSHIP

Former.—"*Section 1.*—There shall be four classes of members: Active, Associate, Senior, and Honorary. Admission to membership in the Association shall be by election. There shall be no numerical limits on any class of membership. Only Active and Senior members shall have the privilege of voting or holding elective office."

Adopted.—"*Section 1.*—There shall be four classes of members: Active, Associate, Senior, and Honorary. Admission to membership in the Association shall be by election. Membership shall be limited, the limits on the respective classes to be determined by the By-Laws. Only Active and Senior members shall have the privilege of voting or holding elective office."

Former.—"*Section 2.*—Election to all classes of membership shall be for life, subject to the provisions of Section 3, following."

Adopted.—"*Section 2.*—Election to Active, Senior, and Honorary Membership shall be for life, subject to the provisions of Section 3, following. After the 1960 Annual Meeting of the Association election to Associate Membership shall be for a limited period of time, as determined by the By-Laws. During this limited period, an Associate Member, if properly qualified, may be elected to Active Membership. At the expiration of this limited period, an Associate Member, if not yet qualified for Active Membership, must either be reelected to an additional period of Associate Membership, or be dropped from the rolls of the Association."

Section 3. Unchanged.

BY-LAWS: ARTICLE III

Former.—"*Section 2.*—There shall be no numerical limit on Active Membership. The candidate, to be eligible, must be a citizen of the United States of America or of Canada, unless in unusual cases this citizenship requirement shall have been waived by the Council. The candidate shall have achieved distinction in the thoracic field, or shall have made a meritorious contribution to knowledge pertaining to thoracic disease or its surgical treatment.

Under usual circumstances, Active Members shall be elected from the Associate Members who have satisfied all requirements for Active Membership. Under unusual circumstances, Active Membership may be awarded to new candidates of outstanding distinction.''

Adopted.—''*Section 2.*—Active Membership shall be limited to three hundred fifty.'' (The remainder of the Section unchanged.)

Former.—''*Section 3.*—There shall be no numerical limit on Associate Membership. The candidate, to be eligible, may be a citizen of any country. All candidates for Associate Membership shall be engaged actively in the practice of clinical medicine, or in teaching, or investigative work in the basic sciences underlying clinical practice, and all shall have demonstrated an active interest in thoracic disease. In the case of those who are engaged in the independent practice of thoracic surgery, it shall be required that they present evidence of sound basic training in general as well as thoracic surgery. Clinicians of outstanding ability, whose interest is limited to a small fraction of the entire thoracic field, may be elected to Associate Membership.''

Adopted.—''*Section 3.*—There shall be no numerical limit on Associate Membership. The candidate, to be eligible, may be a citizen of any country. All candidates for Associate Membership shall be engaged actively in the practice of clinical medicine, or in teaching, or investigative work in the basic sciences underlying clinical practice, and all shall have demonstrated an active interest in thoracic disease. In the case of those who are engaged in the independent practice of thoracic surgery, it shall be required that they present evidence of sound basic training in general as well as thoracic surgery. Clinicians of outstanding ability, whose interest is limited to a small fraction of the entire thoracic field, may be elected to Associate Membership. The limited period of time for Associate Membership, as required by ARTICLE III, *Section 2*, of the Constitution, shall be five years.''

A change which would limit the number of visitors at each meeting was defeated. Thus, the scientific sessions still remain open to those interested and qualified physicians who register in appropriate manner.

The following changes in membership status were approved by the Council and passed by the membership in Executive Session:

Four members were dropped from the rolls of the Association either through the acceptance of their resignations or because of nonattendance.

The following Active members were elevated to Senior status:

J. Dewey Bisgard
Joseph W. Gale
Ernest C. Janes
Clarence A. McIntosh
James L. Mudd
George F. Skinner
George Willauer

The following Associate members were elevated to Active membership:

Thomas G. Baffes
James S. Battersby
Philip E. Bernatz
William S. Blakemore
Ivan W. Brown, Jr.
John C. Callaghan
E. Stanley Crawford
John F. Dammann, Jr.
Julio C. Davila
William W. Heroy
Charles A. Hufnagel
Karl E. Karlson
Harvey W. Kausel
John Lester Kee, Jr.
James Alastair Key
Earle B. Mahoney
Elliott Michelson
Peter V. Moulder
Thomas F. Nealon, Jr.
David C. Sabiston, Jr.
Frank C. Spencer
Lawrence H. Strug
Frederick H. Taylor
William I. Wolff

The following candidates were elected to Associate membership:

Jesse E. Adams, Jr., Chattanooga, Tenn.
Murray N. Andersen, Buffalo, N. Y.
John E. Connolly, San Francisco, Calif.
Milton V. Davis, Dallas, Texas
Hugh F. Fitzpatrick, New York, N. Y.
Joseph J. Garamella, Minneapolis, Minn.
John P. Heaney, San Antonio, Texas
Robert H. Holland, Dallas, Texas
James B. Littlefield, Charlottesville, Va.
Daniel E. Mahaffey, Louisville, Ky.
Charles V. Meckstroth, Columbus, Ohio
Fletcher A. Miller, Minneapolis, Minn.
Thomas C. Moore, Indianapolis, Ind.
Edward R. Munnell, Oklahoma City, Okla.
Joseph W. Peabody, Jr., Washington, D. C.
Robert G. Pontius, Pittsburgh, Pa.
George Robinson, Mount Vernon, N. Y.
Dennis M. Rosenberg, New Orleans, La.
Robert J. Schramel, New Orleans, La.
Thomas W. Shields, Chicago, Ill.
Harold C. Spear, Miami, Fla.
Warren J. Taylor, Boston, Mass.
Milton Weinberg, Jr., Evanston, Ill.
William Glenn Young, Jr., Durham, N. C.